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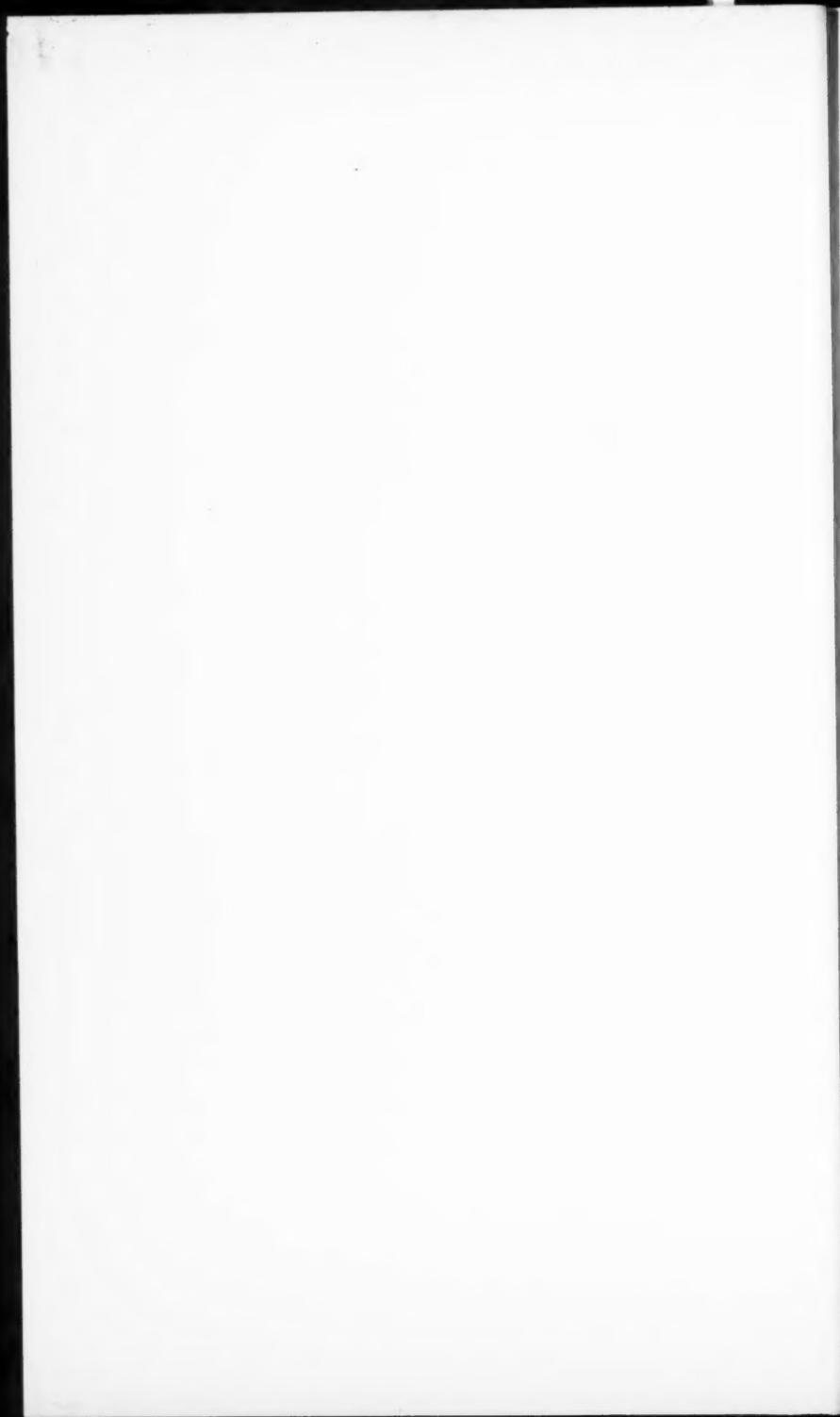
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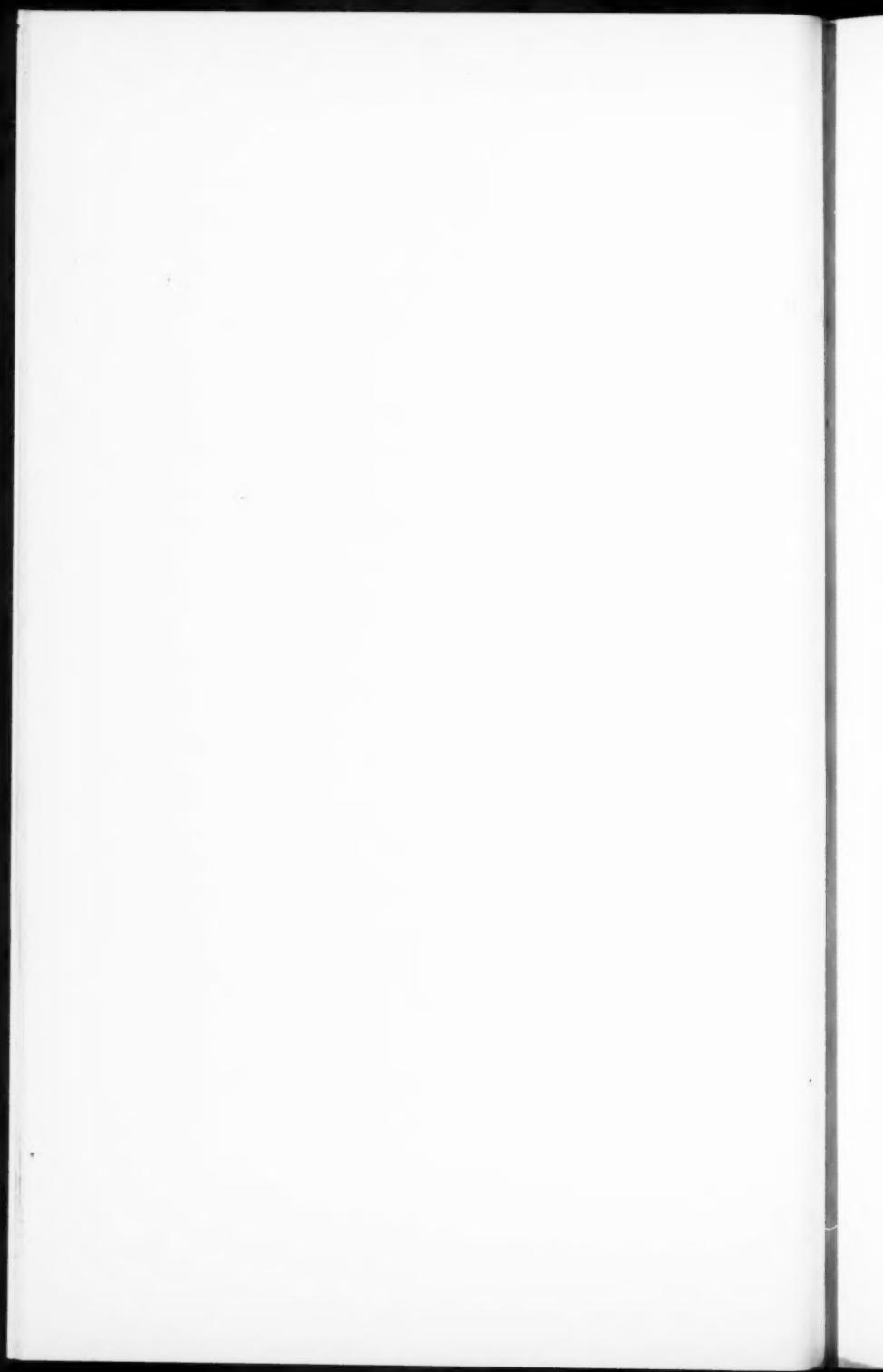
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## PRIMARY CARCINOMA OF THE LUNG. TWO CASES.

**Case I.**—The first patient whom I wish to present today is a man, fifty-six years of age, by occupation a laborer, who came into the hospital coughing very violently, the cough being of a paroxysmal nature.

*Entrance Complaints.*—Cough. This has lasted about three months, but it has been appreciably worse during the past three weeks.

Sputum. He states that he has coughed up about 5 ounces of sputum a day and that this amount of sputum began to appear rather suddenly three months before admission.

Pain. He has no pain when not coughing, but the pain is quite violent, on the right side of the chest only, during the paroxysms of coughing. This has been going on for about two weeks.

Headache. This symptom appeared only during the paroxysm of coughing and has existed especially since the sputum began to be more abundant.

Loss of appetite. He has been losing his appetite gradually and this has been especially marked for the past few weeks.

Loss of weight. He is not very clear about this point, but thinks he has lost approximately 12 pounds in the past month. He is, however, quite sure that he has lost a great deal of strength and his general appearance bears out the two latter statements.

*Onset and Course.*—For many years the patient has been a user of alcoholics, usually whisky or moonshine. His daily consumption varies from one or two drinks daily to a pint or more. For several months prior to the present illness he had been drinking very heavily and he had noticed that he coughed much more each morning on arising, the sputum consisting of mucoid material. Somewhere about this time the loss of strength with a persistent tired feeling, especially when doing heavy work, made itself more and more manifest.

One morning about three months ago he says he felt "very poorly" because of headache and muscular soreness. At this time he coughed considerable blood-tinged sputum. For about a week no more bloody sputum

was raised, and then he had another attack, at which about a teaspoonful of very bloody material was brought up. This condition remained practically unchanged until about two weeks ago, when he began coughing more bloody sputum, also a yellow and at times rather dark tenacious, almost gelatinous sputum. At this time he noticed a marked loss of strength, with great impairment of his appetite, and a moderate degree of pain in the right chest. It was about this time he thinks that he noticed attacks of shortness of breath. During the past two days there has been no especial change in his condition.

*General and Negative.—Gastro-intestinal.*—With the exception of the loss of appetite which has already been noted and a moderate constipation which is in all probability the result of his taking less food, there is nothing worthy of note.

*Cardiovascular.* He does not complain of palpitation or precordial pain or, indeed, of any pain which might be considered as anginal, nor has he had any edema of the extremities.

*Respiratory.* We have inquired, of course, most carefully as to antecedent disease of the lung. He insists that he has never had pneumonia in any form nor influenza at any time. Indeed, he states that this is his first respiratory trouble since he was old enough to remember. He does not suffer at all frequently from sore throats or tonsillitis.

*Genito-urinary.* He denies all venereal infections and disclaims any symptoms relating to the kidneys or bladder. Indeed, he is a rather conspicuous example of a patient who has had few diseases. He remembers only having had measles in childhood. He has never been subjected to any operation.

*Family History.*—The patient is single. His mother is alive and well at ninety. His father died at an advanced age of a rupture and some lung trouble, probably a senile pneumonia. He has two brothers, both of whom are living and well; one brother died of heart disease. Patient knows nothing about there having been any tuberculosis or carcinoma in the family history.

The preliminary examination made on admission yielded relatively little. His temperature was 99° F., pulse 84, quite regular, no inequalities on the two sides, no engorgement of the cervical veins. Respiration was 20 and not especially labored, but during an attack of coughing there was considerable dyspnea. His weight on admission was 196 and it seemed rather clear that he had weighed considerably more, as he is a man of large and powerful build. Blood-pressure was 130/74.

*Physical Examination.*—The salient points are as follows:

Head was entirely negative. Eyes reacted somewhat slowly to light and accommodation, the pupils being midwide, regular, and equal. Ears and nose were negative. Mouth showed teeth in rather good condition for a patient of his social standing. He has had a good deal of dentistry which seemed to be of fair quality. The tonsils which were examined rather closely were small and atrophic, giving no evidences of attacks of tonsillitis. The tongue showed nothing of importance except a slight grayish coating.

Neck. Along the anterior and posterior margins of the sternocleido-mastoid an occasional tiny gland could be felt, not more, however, than i-

frequently the case in normal patients of this sort. The thyroid presented nothing of importance.

Chest. No pulsations were apparent even when searched for carefully in a good light. There seemed to be a slight impairment of the expansion on the right side. The percussion note was stated to be slightly impaired, more especially at the base and middle portion of the right lung posteriorly. Over the entire right side a few moist râles were heard, especially after a paroxysm of coughing. The breath sounds were rather uniformly diminished over the right chest, the whispered and spoken voice being also somewhat diminished over the same area.

Abdomen. The edge of the liver descended about 2 cm. below the costal margin on deep inspiration, otherwise the solid viscera were not palpable. No points of tenderness to be made out.

Extremities. Uppers negative. The lowers showed marked varicosities and old varicose ulcers from the knee downward.

The special senses and cutaneous sensation were intact. The usual reflexes were present and no pathologic reflexes were elicited.

This history was taken by my intern the day after the patient's admission to the hospital and was checked over by my resident. I did not see the patient for a couple of days after his entrance into the hospital. Almost immediately after his admission he commenced to run a little abnormal temperature of a typical septic type, the temperature for the most part going to 100° or 100.5° F. in the evening, and dropping to subnormal in the morning. This has continued up to the present time.

At my first personal examination of the patient, three or four days after his admission to the hospital, I was able to confirm the findings as they were recorded at the time of admission. On discussing the case with my resident it presented itself somewhat as follows: A middle-aged man with absolutely no history of any antecedent respiratory infection develops a cough, which in the course of months becomes greatly aggravated, with a considerable amount of blood-tinged sputum, increasing dyspnea, and some rather anomalous findings in the right lung. One might think of a mediastinal tumor or aneurysm, but the most painstaking examination directed toward these failed to disclose any ground for such a diagnosis. It seemed quite clear that the pathology lay in the right lung and we endeavored in this case as we always do, to come to some tentative conclusion before submitting the patient to fluoroscopy. Going over the right

lung again carefully the following points called for our especial attention: There was definite impairment of expansion, a considerable degree of dulness over the greater portion of the lung, especially posteriorly, this dulness not being as marked as in a pneumonia and yet perfectly clear cut and definite. It was no more marked at the base than farther up. Over this entire area the breath sounds were very feeble and the voice sounds correspondingly weak. Râles were almost entirely absent except that, as above noted, a few moist râles could be heard after a paroxysm of coughing. I think it will be profitable to analyze these physical signs, since, after all is said and done, they are a little out of the usual. The dulness might make us think of a consolidation or perhaps a small amount of fluid, but the diminution of the voice and breath sounds exclude the former, and the very great extent of the dulness, nowhere amounting to an absolute flatness, makes the latter quite improbable. Furthermore, the note lacked that wooden quality with great resistance to the percussing finger that we invariably expect in an effusion of such a size. On the other hand, there was unquestionably a rather extensive pathology in the lung. We might think of a solid tumor in the lung, but even that could hardly produce these signs in the intensity and to the extent in which we found them. On the other hand, this is just the sort of picture that one finds where there is extensive atelectasis and especially when this atelectasis is produced by an obstruction to one of the larger bronchi. This seemed by all odds the most likely and most tenable supposition. We could be practically certain that there was some active process going on in the right lung and with those findings I felt that we could be certain that this process involved one or more of the larger bronchi and that the dulness and diminution of the voice and breath sounds were evidences of bronchial stenosis. That was my diagnosis at the first examination without, however, my being clear as to the underlying pathology.

However, a little reflection will make several points reasonably clear. The patient is fifty-six, has always been an out-of-door laborer, has never had any chronic cough or loss of weight

until this attack, has been free from fever or at least so he thinks until he entered the hospital, and altogether the patient presents a rather different picture than we would expect with the most common pulmonary pathology, namely, tuberculosis. Again, too, the apices are clear and the bulk of the pathology seems to lie somewhere around the hilum at a point where the larger bronchi could be compressed. I do not remember to have ever seen a tubercular process producing a bronchial stenosis, unless perhaps in some small measure in a very old fibroid type of this disease. This the patient certainly has not. However, it is an old rule which says that one should consider the common things first, and so my first direction was to make a careful study of the sputum, both by straight smears and by the Löeffler antiformin method for tubercle bacilli. This was repeatedly done with negative results. Cultures of the sputum were made also, with negative results. About this time the character of the sputum changed quite materially and took on a jelly-like consistency, very homogeneous and of about the color of currant or grape jelly. On inverting the cup and even vigorously shaking, the sputum refused to be dislodged. Examination for blood shows that this reddish color is due to a very homogeneous distribution of blood pigment. I personally am quite skeptical about any particular kind of sputum being invariably found with a given pathologic process, but one could not help being impressed with the fact that this was a perfectly typical specimen of the currant jelly sputum which has been for a half century supposed to be characteristic of neoplasms of the lung. This sputum coupled with the fact that no evidences of tuberculosis could be found directed our attention very definitely toward the possibility that we were dealing with a pulmonary neoplasm.

Now what things can be done to establish a diagnosis? Sometimes a bronchoscopic examination when made by expert hands may reveal a carcinoma if this be of bronchogenic origin. Such an examination was made and indeed made twice, but was not very satisfactory and yielded no data of diagnostic value. The various laboratory examinations were also unproductive of data which could help. The blood-count was 4,200,000, with

11,000 white cells, with the usual distribution of the various types of cells. The urine was negative although examined repeatedly. In spite of the fact that the patient gave absolutely no complaints relating to his bladder, a careful prostatic and bladder examination was made, but everything was quite negative. The cultures of the sputum showed the ordinary organisms. The blood chemistry was quite negative and the blood Wassermann and Kahn test disclosed nothing abnormal. Examination of test-meals and stools was quite negative. We had, therefore, no reason to diagnosticate any tumor, such as a carcinoma of the prostate or pancreas, or a hypernephroma which might give possibly metastases in the lung. An *x*-ray examination was now made, with the following result:

"The films show marked increase of the right hilum shadow with extension into the upper lobe. The condition may represent an old healed pulmonary tuberculosis."

At the same time the roentgenologist called attention to the possibility of a carcinoma which, of course, we had already been considering.

From the time of admission up to the present a half-dozen sets of films have been made, and it is interesting to note that the atelectasis of the right lung, which was recognized clinically almost immediately after his entrance, was present for some time before it could be recognized by the roentgen examination.

At the present time what progress have we made toward a more certain diagnosis? I think we can state quite positively three or four things: First, there is a definite stenosis of either the primary bronchus on the right side or of several of the larger branches. Second, tuberculosis is to be excluded. Third, we have no diagnostic data on which to base an assumption of any of the very rare conditions in the lung, such as actinomycosis or hydatids. I once had the opportunity of diagnostinating an echinococcus cyst of the right upper lobe, but these cases are of extraordinary rarity. I have never seen but that one case in the lung except as an extension from a liver echinococcus. In that case the diagnosis was made by the presence of the characteristic membranes, in large numbers, in the sputum.

This leaves us with almost the necessity of making the diagnosis of a primary neoplasm of the lung unless there be a primary mediastinal tumor with metastases in the lung. I have already stated why I do not believe that the primary tumor was in the mediastinum, and so I think I am ready to go on record as saying that I think in all probability we have to do with a primary neoplasm of the lung probably taking its origin from one of the larger bronchi. I make this latter statement because from the very beginning of the history everything pointed to the right lung and a copious and characteristic sputum has been present for a long time and in considerable amount, defying all treatment.

Perhaps a few words on the subject of carcinoma of the lung may be in place.

Of course it is perfectly well known to you that, in general, metastatic tumors of the lung are as common as primary tumors are uncommon. Personally and without any regard to pathologic statistics the majority of lung tumors which I have seen have been metastases from hypernephromata of the kidney and occasionally metastases from testicular tumors. One may find occasionally a carcinoma, which we could rather expect in this patient in view of his age, following a thyroid or an esophageal carcinoma. The primary carcinomas of the lung are said to occur in about 1 per cent. of all carcinomata, though my personal opinion is that they are more common. I have seen several cases in the past year and there is a probable case in addition to the two I am showing you on the floor at this time. The majority seem to be bronchogenic in origin, in other words, taking their beginning from the epithelium of the bronchi or perhaps from the alveoli themselves. In this instance in view of the comparatively early occlusion of the bronchus, it would seem highly probable that the diagnosis of carcinoma, if correct, would almost certainly mean that its origin was in one of the larger bronchi. I have seen several cases of carcinoma arise in old tubercular cavities or at least such was the explanation of the pathologist.

One sees statements constantly made that the diagnosis of carcinoma can be made by finding of so-called cancer cells. I

confess to a good deal of skepticism on this subject, although I have seen the diagnosis made by coughing up pieces of tumor material, but in most instances they have been so necrotic as to make the histologic examination a matter of great difficulty. This was the case in a patient about a year ago. There is one thing else which we can do which may shed some light upon the diagnosis. It not infrequently happens that there is a little bit of fluid in the pleura and it might be that the characteristics of this fluid would shed light upon the diagnosis. Again, one can make a diagnostic puncture of the lung itself, although this is not at all free from danger, and particularly so in this case, since we would have to go in pretty deep and pretty close to the great vessels to strike the tumor mass.

Summing up the case then as a whole, we are probably dealing with a bronchogenic carcinoma taking its origin from one of the larger bronchi of the right lung. If our diagnosis is correct, the case will, of course, go on to the usual termination. It may be that cavitation will develop, in which case we would naturally expect the sputum to become purulent and fetid as a result of secondary infection. I will show you the case at our next meeting or at least some time in the near future and report the progress.

*Three Weeks Later.*—Most of you will remember the patient whom I presented in this same clinic three weeks ago. We did not come to a definite diagnosis at that time, but we considered a number of things and, in particular, a primary carcinoma of the lung. Let me recall to your mind that at that time the patient, who had been fever free for the first few days after admission began to develop a temperature, running up as high as 100.5° F. in the evening. During the past three weeks this has kept up and the temperature range has become steadily higher and reached 101.5° F., sometimes 102° F., every evening, and dropped down to subnormal every morning. This, of course, bears all the earmarks of a septic temperature. The leukocyte count has gone up from 11,000, when he first came in, to between 17,000 and 20,000 at the present time. The sputum has been increasing steadily in quantity, and, needless to say, many examinations for tubercle

bacilli have been made but no organisms found. Cultures have shown repeatedly *Staphylococcus aureus*, diphtheroids, some fusiform bacilli, a few colonies of pneumococci and *Streptococcus viridans*. The patient has lost weight steadily and progressively. Numerous x-ray examinations have been made without very much change being noted from the first examination, except that the density extending into the right lower lobe is increasing steadily. Our x-ray colleague is now of the opinion, as we are, that we have to do with an infiltrating type of carcinoma of the right lung.

A few words about the differential diagnosis may again be in place. One might consider the possibility of primary syphilis of the lung, but in my judgment this is, under the conditions before us, extremely improbable. In addition to the inherent rarity of this disease in the adult in the absence of other signs of lues, we have a negative Wassermann in our patient. The sputum shows none of the findings which occur in echinococcus of the lung, of which I have personally seen but one case, but in this case the membranes were so characteristic as to make the diagnosis very easy. Besides this patient has been nowhere where an echinococcus infestation would be at all probable. On the other hand, it is plainly evident that we have before us a patient that is rapidly going downhill and is gravely ill. In view of all these findings I think we may now venture the diagnosis with a reasonable degree of certainty and say that we have a primary carcinoma of the lung.

*Subsequent Course.*—I am showing you today the autopsy material from the patient I presented in clinic a number of weeks ago and in whom we made the diagnosis of primary carcinoma of the lung. His death occurred yesterday after a stay of three and one-half months in the hospital. A short time after I showed the patient to you a rather unexpected event occurred. His sputum became much more profuse and more foul smelling, which went hand in hand with the septic temperature and which we attributed to a secondary cavitation in an occluded bronchus. One day my intern noticed that there was quite a sizable piece of tissue in the sputum, and he did the eminently correct thing,

to take it down to my colleague, Professor Jaffe, for embedding and sectioning. A few days later Professor Jaffe reported verbally that the bit of tissue was only slightly necrotic and that it showed unmistakably the structure of a carcinoma, and that under the circumstances his belief was that we were dealing with a primary carcinoma of the lung.

*Summary of Autopsy Findings.*—I think I can do no better than to read you the summary of the autopsy findings which you can verify for yourself.

Lungs: A flat papillary growth present inside the right bronchus at its bifurcation, encircling it and almost occluding the lumen. It extends for 3 cm. into the bronchus. The larger bronchi of the right upper lobe are surrounded by white tumor tissue constricting the bronchi very markedly. The entire bronchial tree on the right side as far as the finest bronchi is filled with thick mucopurulent material. Numerous tiny abscesses are present in all the lobes on the right side, the entire lung being filled with pus.

The right peribronchial lymph-glands form a mass 5 x 8 cm. of white tumor tissue pressing on the bronchi.

Anatomic diagnosis: Bronchogenic carcinoma of the right lung, with extension to the right bronchial tree and obstruction in the right bronchus. Multiple abscesses in the right lung and in the left lower lobe. Confluent bronchopneumonia of the right lower lobe. A few metastatic carcinomatous nodules in the liver.

Little remains to be said about this case except that it seems wise to call your attention to the fact that the fever was, as we had premised, in all probability due to the secondary infection and not to the carcinoma itself, and this explains the varying statements with reference to the temperature which are current in the literature on the subject.

**Case II.**—The second patient is a man seventy-four years of age, a native of Belfast, Ireland, who comes into the hospital apparently not acutely ill and with the following:

*Present Complaints.*—He has had a cough for the past three months. This cough, he thinks, was no different from the cough of an ordinary cold except that it has been persistent and associated with considerable expectora-

tion, which has amounted to a half cup daily. Associated with this has been considerable pain in the right side of the chest and in the back. This pain is distinctly worse on coughing. He thinks the pain started at about the same time as the cough. He has lost a good deal of weight, he thinks perhaps 30 pounds, and he attributes this to his loss of appetite due to the continuous coughing.

*Onset and Course.*—As already indicated, the patient "took a cold on his lungs" about three months ago. He did not go to bed, although he coughed even then considerably, especially at night. From the very beginning he had sputum of a white mucoid nature and perhaps a half cupful daily. He has never expectorated any blood. He began to lose weight and strength, although the appetite was relatively well preserved. The patient had a dull ache in the right chest, rather diffuse in type, worse after coughing, but not materially affected by breathing. He does not think he has ever had fever in the afternoons. During these three months he did not attempt to work, but has been around and about ever since.

Patient thinks that the condition has gradually become worse up to the time of his admission to the hospital. He complains bitterly of loss of sleep owing to the insistent cough. The sputum has not changed materially. His present weight is 117 pounds and he weighed 152 a year ago.

*General and Negative.*—Up to the time of this sickness his appetite had always been good. He had never had nausea, vomiting, or any other symptoms referable to the stomach. His bowels had been regular and there had been no diarrheas.

*Cardiovascular.*—On being asked, he thinks he has had an occasional attack of tachycardia, but no palpitation, cyanosis, shortness of breath, or edema.

*Respiratory.*—Up to the onset of the present condition he showed no symptoms pointing to the respiratory tract, in particular no pain, expectoration, or cough.

*Genito-urinary.*—The only symptom of consequence complained of in this connection is that of nocturia. He has to get up four or five times a night, and we may dismiss this by saying that it is due to an enlarged and hypertrophied prostate. I may anticipate a little by saying that a careful examination of this organ reveals no evidences of tumor formation.

*Nervous.*—There are no especial neurologic symptoms. The special senses show only the changes due to his age.

*Past History.*—This is entirely negative and he has had no severe diseases at any time in his life. He has never been operated on, except that he had both legs broken many years ago for which he received some surgical treatment.

Venereal history is negative.

*Family History.*—Shows nothing worthy of note.

*Physical Examination.*—I will give you the salient points of the physical examination as they were described on his entrance to the hospital.

The patient was thin, poorly nourished, evidently somewhat short of breath, since he was lying in bed with a back-rest, and could not take the recumbent position comfortably. He was coughing frequently. The head,

eyes, ears, nose, and throat showed nothing especial except the usual bad teeth found in patients of this social class.

The cervical glands were not palpable.

*Chest.*—This was somewhat emphysematous in type and showing, of course, the atrophy due to age and the loss of weight. The clavicles were very prominent. Inspection showed that the expansion was definitely limited on the right side with a normal excursion on the left. The tactile fremitus was increased over the right side both front and back. On percussion a dull note was found more particularly over the upper and front portion of the chest, becoming flat at about the level of the nipple. Auscultation showed that the breath sounds were somewhat bronchial but distinctly distant on the right side. The spoken voice showed a somewhat increased transmission over the right lung, especially at the base.

*Heart.*—Apex-beat in its normal position. The boundaries showed no changes and the sounds were all clear.

*Abdomen.*—Somewhat "pot bellied," but otherwise showed nothing of consequence, in particular no masses could be felt nor was any fluid demonstrable in the flanks.

The extremities showed the usual thin, emaciated appearance. No edema present.

*Rectal examination* disclosed nothing unusual with the exception of a moderate enlargement of the prostate.

If we attempt to picture to ourselves a diagnosis with these symptoms alone we should have a pretty difficult time. About all that we could say was that it seemed obvious that the man had some severe intrathoracic condition, presumably in the lungs or mediastinum, since nothing abnormal could be detected in the heart. I did not see the patient immediately on his admission, but I find a note several days later to the following effect: "Patient is sitting up in bed in considerable respiratory distress. Physical examination reveals the same findings as on admission. There is now dulness amounting almost to flatness over the base of the right lung, with a few medium-sized râles. It is especially noted that the breath sounds were well transmitted."

In the meantime considerable laboratory work had been done upon the patient, the most important of which I will read. The blood showed 3,100,000 reds; 58 per cent. hemoglobin, with a leukocyte count of a trifle over 6,000 and the differential count substantially normal. No abnormal cells were found in the stained specimen.

Urine was entirely negative. The phenolsulphophthalein

renal test showed a considerable diminution in the output, this amounting only to 45 per cent. in three hours. A Mosenthal test was made, but there was no fixation of the specific gravity, no increase in the night urine, and in general the outcome of this test of the kidneys was that they were in fair condition. The blood-sugar was 91 mgs. and NPN 29 mgs. per 100 c.c. of blood, both of these of course being well within normal limits. In spite of a persistent denial of any venereal infection, a 4+ blood Wassermann was found.

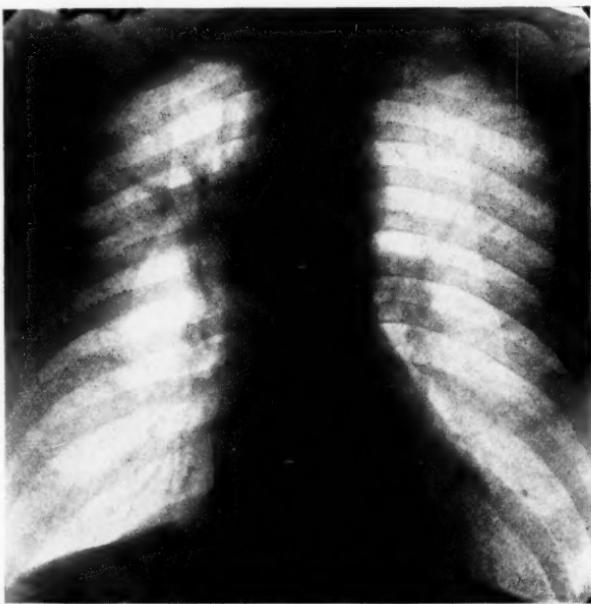


Fig. 1.—Primary bronchogenic carcinoma of lung. Early stage.

*Roentgen Examination.*—In view of the fact that our physical examination pointed pretty clearly toward intrathoracic pathology, we are especially interested in the results of the x-ray examination of the chest. I will read you the result of this.

"Fluoroscopic and film made in postero-anterior direction

showed diffuse increased density over the right lower lobe and mottled density above this, reaching to the level of the second rib. Roentgen diagnosis: Probable pleural effusion on the right side and intrathoracic pathology."

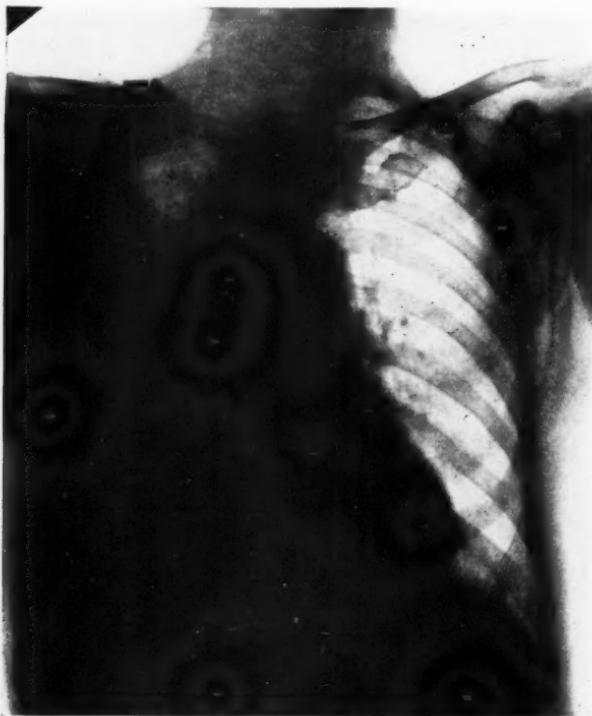


Fig. 2.—Primary bronchogenic carcinoma of lung. Late stage.

Needless to say this roentgen diagnosis did not help us much except that it confirmed our idea that there was some trouble in the thorax and probably in the right lung. A little reflection at this time enabled us to take at least one or two steps farther forward in the diagnosis. Numerous specimens of sputum had been examined, but failed to show tubercle bacilli, even when examined with the Löffler concentration method. We have not

been hopeful of finding bacilli since a tuberculosis is very unlikely to develop at this age and since the unilateral findings in the lung were hardly those of any ordinary tuberculosis. Furthermore, the patient had had no fever during his stay in the hospital and this, of course, militates very strongly against the diagnosis of tuberculosis. Repeated physical diagnoses seem to rule out aneurysm and mediastinal tumor, especially the mediastinal type of Hodgkin's disease.

The next thing to be thought of at this time seemed to be that we might have a metastatic growth in the right lung secondary to some neoplasm elsewhere. In particular the prostate was thought of, but, as I have already stated, the examination of the prostate gave no ground for such a suspicion. The gastrointestinal tract was not examined by means of test-meals, since the patient seemed really too sick to subject him to such a procedure in view of the entire absence of symptoms pointing to the stomach or bowel. However, the stools were repeatedly examined for blood with negative results. In view of the very great frequency with which kidney tumors, especially hypernephromata, metastasize in the lungs, especial attention was paid to the search for blood in the urine, which, of course, is one of the most frequent diagnostic points of a hypernephroma. This was always negative. Perhaps it may strike you as a little far fetched that I should be discussing the possibility of a metastasis in the lung when all the symptoms point only to the lung and when there was no complaint of trouble elsewhere. I can assure you that this is not so, since it has undoubtedly happened to every clinician of experience to have a lung metastasis crop up as the first symptom noted in a kidney tumor. We had an autopsy on such a case only a short time ago. The same thing applies occasionally to testicular tumors. Since repeated and careful examination failed to find any evidences of a malignant tumor, particularly a carcinoma anywhere else, we commenced to think of the possibility of a primary carcinoma of the lung. What evidence might be adduced in favor of such a diagnosis? To begin with, there is the age of the individual, the very definite intrapulmonary pathology, the constant pain, the per-

sistent cough with expectoration of a moderate quantity of sputum, in the absence of any evidence of bronchiectasis or tuberculosis. The absence of fever points distinctly away from tuberculosis. I should like to call attention to the fact that during his stay in the hospital the sputum underwent a very marked change, becoming very thick and jelly-like, but remaining fairly clear and mucoid. As you see, although this sputum cup is half full it can be turned upside down without the sputum spilling.

We decided about ten days after the first *x*-ray of the chest to have a second one made, which was done, with the following result:

"Fluoroscopic examination showed a wedge-shaped area extending outward and upward from the right hilum region, which looked like an intralobar pleural effusion. Above this the lung showed irregular density and there was an area of relative radiolucency below and external to it. The film made showed practically the same findings as the previous film."

It has undoubtedly not escaped you that the physical findings described did not fit in well with the idea of a pleural effusion, but rather with that of a collapse of the lung or atelectasis. To settle this matter we decided to make an exploratory aspiration.

I hardly expected to find any fluid, and I determined in view of the possibility of a primary carcinoma of the lung that, if I did not get any fluid, to take a very thick needle and to attempt to puncture the tumor mass itself. I am perfectly aware that this is not always a safe procedure, but a study of the film seemed to show that a part of the tumor was relatively near the surface and might be punctured by a needle. We actually carried out the proposed puncture, using a rather thick needle of large caliber and using considerable suction. We succeeded in getting a few drops of bloody material looking like tissue juice. This was immediately sent to Professor Jaffe with the request that a careful study be made of this material to see if any cell complexes could be found which might substantiate our tentative diagnosis of carcinoma. A report on this material made a

few days later stated that many cells undergoing mitosis and especially irregular mitosis had been found and that at the very least it was highly suspicious of a carcinomatous condition. A week or so later, the symptoms and signs remaining absolutely unchanged, a few small axillary glands could be felt and it was determined to remove one of these for a biopsy. This was done and the pathologic report came back in due season to the effect that it was undoubtedly a metastasis of an adenocarcinoma in a lymph-gland.

With the results of these examinations we are emboldened to make the diagnosis of a primary carcinoma of the right lung. I think all the physical signs can be adequately explained on this assumption. The pain, the absence of fever, the progressive and insistent cough, the thick gelatinous tenacious sputum, without blood, the absence of tubercle bacilli, the progressive emaciation and cachexia all fit in perfectly with this diagnosis. My interpretation of the findings in the right lung is that we have a considerable area of the lung atelectatic as a result of a practically complete occlusion of a bronchus.

If one stops to consider a moment it will be evident how a primary carcinoma of the lung may produce widely divergent symptoms and physical signs, depending on the location, on the relationship to the larger bronchi, and to a very considerable extent upon the number of local metastases and perhaps also a secondary infection. Let us consider these points one by one. Let us suppose that the tumor arises from one of the medium-sized bronchi pretty much in the center of the lung. Under these conditions, with no larger bronchus occluded the tumor may attain a very considerable size with but little pain, no fever, and perhaps little or no cough. After a while local metastases, especially in and around the hilum of the lung occur, and these give rise to a severe cough reflex and may, in their turn, produce an occlusion of one of the larger bronchi. This occlusion if anywhere nearly complete gives rise to an atelectasis with, of course, dyspnea and persistent irritative cough. In my judgment fever is very variable. I have seen several cases in which fever was marked very early and other cases, such as this, in which fever

was absent. If one looks through the literature one will find one set of authors stating that the absence of fever is quite characteristic and other authors declaring that fever is directly of diagnostic value. It seems to me that both of these statements may be correct and that the explanation is to be found in a secondary infection with stagnation of the sputum in bronchiectatic cavities which develop as a result of the occlusion of bronchi. I think this also explains the different character of the sputum. The older authors laid especial stress upon the presence of raspberry-jelly-like sputum which means nothing more or less than that there is a persistent admixture of small quantities of blood. This again is entirely accidental, since the location of the tumor decides whether or not small vessels will be eroded. In my judgment it is the constant daily occurrence of considerable quantities of sputum of a thick gelatinous character, showing no tubercle bacilli and uninfluenced by treatment that is especially characteristic.

Perhaps some of you may have been influenced sufficiently by the finding of a 4+ Wassermann to think of the possibility of syphilis of the lung. My own experience is that primary syphilis of the lung in the absence of manifest late syphilitic lesions elsewhere in a patient of this age belongs to the very greatest of rarities. I have seen personally many more cases of primary carcinoma of the lung than of syphilis of the lung of this character. Of course, I am not referring at all to the syphilitic lesions which occur in the lung as the result of hereditary infection. I may say parenthetically that the patient has been placed on vigorous antisyphilitic treatment, but my judgment is that it is most unlikely to yield any useful results.

To sum up this case then, we feel that we are justified in making a reasonably positive diagnosis of primary carcinoma of the lung which, in view of its location, is probably bronchogenic in origin. The histologic diagnosis of a carcinomatous glandular metastasis is quite difficult, but in view of the expert pathologic help which we have had, I have every confidence in its correctness.

*Subsequent History* (May 31, 1927).—The subsequent course

showed nothing radically different from that which had already been sketched. The patient grew progressively more and more emaciated and the cough and sputum remained just as before, and there was at no time any abnormal increase in the temperature. Indeed, temperature has been conspicuously absent during the two months of his stay in the hospital. He died of progressive exhaustion and weakness. An autopsy was fortunately obtained and a most careful gross and microscopic examination was made of the tumor and the various organs.

*Summary of Autopsy Findings.*—The right lung firmly attached to the wall; pleura transformed into a firm plate 3 to 10 mm. thick, of whitish color, with opaque yellowish lines and specks. Diffuse infiltration of the anterior lower portion of upper lobe, which on cutting appears finely granular and of pale yellowish-gray color, with irregular whitish areas. Whitish medullary masses have infiltrated the muscles between the right upper ribs and the diaphragm. They are also found in the posterior wall of the chest where they have caused some erosion of the transverse processes of the upper dorsal vertebrae.

*Anatomic Diagnosis.*—Medullary carcinoma of the right upper lobe with infiltration of the pleura. Metastases to the tracheolymph-glands, to the right adrenal, to the kidneys (very small). Brown atrophy of the heart and liver. Atrophy of the spleen. Granular atrophy of the kidneys.

It will thus be seen from the anatomic diagnosis that our diagnostic considerations were solidly founded.

#### SUMMARY OF DIAGNOSTIC FINDINGS OF THE TWO CASES

There remains but little to be said that has not already been said in the discussion of these two cases. It is well to remember that about 1 per cent. of all carcinomata are primary in the lung. The symptoms which call for particular investigation are pain, persistent cough not otherwise explained, persistent sputum of a thick gelatinous character, which may or may not be intimately mixed with blood. The physical findings are uncertain and depend upon the location of the tumor. If a bronchus is occluded the signs of atelectasis of the part of the lung supplied by that

bronchus may be found. A bronchiectatic cavity may develop as a result of secondary infection above the occluded area. Fever may or may not be present, depending probably upon the degree of secondary infection. Various pressure symptoms pointing to mediastinal involvement may develop from metastases in the mediastinum. A pleurisy, generally of a hemorrhagic nature, occurs in many instances. In these 2 cases we have been extremely fortunate in being able to make the diagnosis practically certain by finding in the sputum of the first case a bit of tissue from the tumor which could be recognized as carcinomatous and in the second case, with less certainty, by the examination of tissue juice and a small lymph-gland. Although I have had the opportunity of seeing a considerable number of cases of primary carcinoma of the lung, in only one or two of them have I had the good fortune to be able to confirm the diagnosis from the sputum. It is distinctly unusual to be able to make the diagnosis in this way.

CLINIC OF DRs. ISAAC A. ABT AND  
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### SIGNIFICANCE OF LYMPHOCYTOSIS IN INFANTS AND CHILDREN

LYMPHOCYTOSIS is frequently observed in a variety of conditions in young childhood during health as well as disease. Physicians and students alike are frequently puzzled by the increase of the lymphocyte elements in the blood. The question is frequently asked "what is the normal lymphocyte count in infants and children?" also "when is a lymphocytosis normal and under what circumstances does an increase in these blood elements constitute disease?" In order to answer these questions more satisfactorily we have undertaken a study of this subject and are presenting a review of the literature without attempting any analysis of our own material. Occasionally, a carefully prepared review has a definite value for students and practitioners, because the work of many authors is selected and assembled. This is the problem which we shall attempt in this clinical presentation.

The lymphocytes constitute about 20 to 25 per cent. of all the leukocytes contained in the blood. They are about the size of erythrocytes, contain a large nucleus, and have a very small quantity of protoplasm. The nucleus is round to oval in shape, is fairly rich in chromatin, and has an easily recognizable nucleolus. Large lymphocytes occur in considerable number only in pathologic conditions. Lymphocytes are thought to have their origin in the lymphatic system, especially in the lymph-nodes and lymph-follicles of the spleen.

In children the number of lymphocytes is physiologically much greater than in adults. The lymphatic apparatus is

also much more developed and reacts more readily and more extensively to disease processes, irritants, and to infectious agents.

It is recognized now that the number of lymphocytes in a given quantity of blood is variable, though if they be present in excess we conclude that there is an increased activity in the lymphatic tissues. Conversely, if they are decreased in number we infer that there is a diminished activity in the lymphatic tissues. Consequently, we may speak of hyper- or hypofunction of the organs which are supposed to produce the lymphocytes.

It is generally recognized that in childhood relative lymphocytosis is a physiologic condition. Perhaps this may be explained by the fact that during the first period of life several of the lymphatic organs are of maximum size and function to a high degree. We know that the spleen, thymus gland, tonsils, and other lymphatic structures reach their greatest size and activity during this period of life. It is also known that children show a relatively high lymphocytosis in certain infections, while if the same processes occur in adults no similar alterations are observed in the hemogram.

Lymphocytosis may be observed in measles, scarlet fever, mumps, anterior poliomyelitis, and ringworm. Several authors have called attention to the fact that lymphocytosis may occur in mountainous regions.

The function of the lymphocyte is variable and it may serve a variety of purposes, depending upon the presence of certain specific stimuli as well as upon the influence of external factors on the patient. Under certain circumstances the lymphocyte assumes the function of a phagocyte or it may assist in the digestion of fat; or it may become a fibroblast, or it may act as an agent which neutralizes certain bacterial toxins in the alimentary tract.

Small lymphocytes are not only phagocytic, but they also play an important part in the defense of the body against micro-organisms. Bartels observed that the virulence of tubercle bacilli was decreased if they were mixed with lymphocytes before injection into animals.

According to Bergell, lymphocytes may act as macrophages

which ingest dead cell material, leukocytes, and pigment, and under certain conditions take up bacteria, especially in chronic infectious diseases such as tuberculosis and leprosy. On the other hand, these cells are rarely phagocytic to streptococci and staphylococci.

**Normal Cell Count. Variations in Health.**—It is important that we should establish a standard for the normal number or percentage of lymphocytes at the various periods of life. It will be recalled that Ehrlich originally estimated that lymphocytes constituted 20 to 25 per cent. of the total number of leukocytes.

It has been shown that in the tropics 30 to 40 per cent. of lymphocytes is a normal count. In the newborn there may be 50 or 60 per cent. of lymphocytes, though of these, 15 per cent. may be large mononuclear cells.

With increasing age the number or the percentage and the absolute number of lymphocytes tends to diminish during health, though at the tenth year of life one may find 40 or 60 per cent. in healthy children. From the tenth year on, lymphocytosis as a rule diminishes more rapidly, though marked individual variations may occur. The reaction of the lymphatic system to infection or other irritants is more marked in the child than in the adult. This is observed particularly in typhoid fever from the tenth to the fourteenth year of life. In this group and under these conditions lymphocytosis is more marked than in the typhoid fever of later life.

Lymphocytosis is sometimes observed during childhood without being able to demonstrate any pathologic changes. This is frequently interpreted by hematologists to indicate active resistance on the part of the body against infections or to show the reparative function of the organism after infections. At any rate it is thought to indicate that the regenerating power of the blood-making tissues and organs are at their highest power and efficiency. Indeed, so frequently is there an increase in the number of lymphocytes, with a corresponding activity of the lymphatic organs, that the condition has been spoken of as *physiologic lymphatism*.

Various authors have remarked on the fact that an exclusive carbohydrate diet will increase the lymphocytes at the expense of the leukocytes, while others have shown that an excessive fat diet raises the number of neutrophilic cells.

Other authors think that the excessive ingestion of fat as well as carbohydrate may produce lymphocytosis. Indeed, it appears that in healthy individuals on a one-sided diet of carbohydrate and fats there is a greater increase in lymphocytes than when the diet contains an excess of proteins.

After physical exertion it has been observed that the total number of lymphocytes increases rapidly. The number may be doubled after ten minutes' exercise.

Lymphocytosis is commonly observed during whooping-cough, indeed, it is commonly regarded as pathognomonic for this disease. Ehrlich believed that the lymphocytosis was due to enlargement and increased activity of the bronchial glands. But it has also been pointed out that the increase is greatest during that period of the disease when the paroxysms are most severe. This leads a number of writers to believe that the lymphocytosis of whooping-cough is due to the agitation and exercise which results from the coughing paroxysm. Occasionally, however, lymphocytosis may be observed early during the course of whooping-cough, especially in young infants.

Sometimes a very definite lymphocytosis is observed after prolonged crying and the same condition may result after a protracted epileptic seizure.

The fact has already been referred to that under certain circumstances lymphocytosis indicates the struggle of the organism against infection and is of favorable prognostic significance.

During septic and other infectious processes a high lymphocytic count is regarded as a protective process and is a favorable prognostic sign. During the post-infectious period lymphocytosis may be interpreted as a condition which indicates that the toxemia produced by the infection has been more or less overcome, though it is a clinical observation that high lymphocytosis may occur in septic infections. It has been suggested that in some cases lymphocytosis might be explained by an abnormal reac-

tion of the lymphatic apparatus with weakness of the neutrophilic system. H. L. Tidy reviewed a large number of cases in which it had been claimed that sepsis was associated with lymphocytosis. While he thinks where lymphocytosis occurred the condition was either acute leukemia or the so-called glandular fever, we have seen children die of acute septic infections with purpura which simulated acute lymphatic leukemia. The septic infection was confirmed by blood-culture and autopsy findings.

J. M. Read is inclined to divide lymphocytosis into two groups according to the mechanism of production. In one, a true lymphocytosis takes place which is associated with hyperplasia of the lymphatic structures of the body. In this form an actual increase in the total number of large and small lymphocytes takes place. There is another form which Read regards as a pseudolymphocytosis. He thinks this type is caused by mechanical forcing of the young cells into the circulation from the recesses of the spleen and other lymphatic structures.

In those cases where the disturbance in function of the lymphocytic or myeloid system occurs, young immature cells are found in the blood; this indicates that these organs are not functioning properly and are no longer able to retain these cells until they have come to maturity, or to prevent the immature cells from gaining access to the circulation. This condition is observed in German measles where lymphoblasts are found in the blood, indicating that the German measles has disturbed the function of the lymphatic organ.

It is a well-known fact that in this disease disseminated glandular swelling and enlarged spleen may cause an increase in the lymphocytes and produce a hemogram which may resemble acute leukemia.

In the pneumonias of childhood lymphocytosis is frequently observed, whereas in adults the same infection causes an increase in the polymorphonuclear cells. A similar blood condition is observed in typhoid fever where a lymphocytosis is frequently observed in the later stages of the disease.

So much has been written recently of the infectious mono-

nucleosis of childhood that only a word need be said concerning this condition. Within the past few years it has been pointed out that many of the so-called cases of glandular fever or Pfeiffer's disease are characterized by the occurrence of a relative and absolute increase in the mononuclear cells of the blood. This condition was first described by Cabot in 1913, though Türk described mononucleosis in this peculiar form of adenopathy in 1907, and it is interesting to note that this author interpreted this monocytic reaction as an example of acute lymphatic leukemia, but after following up the patients he found the condition to be a disease *sui generis*.

Marchand reported another series of cases with mononucleosis in 1913. Glandular fever or infectious mononucleosis is an acute febrile disease which is characterized by marked lymphocytosis, enlarged lymph-glands, and spleen. The disease usually ends in recovery. These cases may resemble acute leukemia. The disease occurs in every period of life, though children and young adults are most commonly affected. Tonsillitis, pharyngitis, and nasopharyngitis usually precede the onset of the attack and subsequently the glands enlarge, sometimes to considerable size. Superficial and deeper glands may be involved. The important fact about this illness is that it may be confused with more serious and fatal diseases, though glandular fever almost invariably ends in recovery.

A peculiar form of pharyngitis and tonsillitis has recently been described in young people up to the thirtieth year. The fever lasts longer than is usual in the ordinary sore throat and is associated with considerable enlargement of the lymph-nodes, liver, and spleen.

On superficial inspection the condition may resemble diphtheria. The blood-picture is characteristic. Monocytes are markedly increased, sometimes to 70 per cent., though the usual number averages 20 to 40 per cent. This monocytosis may persist for weeks or months. The lymphocytes and neutrophils are correspondingly decreased in this condition. The lymphocytosis may be so markedly increased that an acute lymphatic leukemia may be suggested.

Lymphocytosis frequently occurs in the later stages of infectious diseases, and for this reason may be spoken of as a post-infectious lymphocytosis. On the other hand, lymphocytosis may occur relatively early in some of the infectious diseases, and this is to be particularly noted in typhoid fever. In this disease the lymphocytes may exceed the neutrophils, and no less an authority than Nägelei considers this as evidence of an immunity reaction. It has also been pointed out that the simultaneous occurrence of leukopenia and lymphocytosis may be regarded as a diagnostic blood finding characteristic for typhoid fever. It has also been said that the severer the attack of typhoid, the more marked the leukopenia and the more marked the lymphocytosis. An increase in lymphocytes is frequently observed after injection of typhoid vaccine. If complications are about to occur in typhoid fever patients or if an unfavorable course of the disease is taking place the lymphocytosis may be arrested and polymorphonucleosis occur.

In chronic malaria a definite mononucleosis and lymphocytosis may be observed. The leukocytes often carry pigment in chronic malarial infection. It is also of interest to note that lymphocytosis increases as the malarial attack tends to abate or diminish.

Tuberculosis of the lymph-glands may cause an increase of the lymphocytic cell in the early stages of the disease before extensive destruction of the lymph-nodes has occurred.

After the lymphatic structures have undergone degenerative changes lymphopenia ensues. The same condition holds in any form of severe tuberculosis, namely, a diminution in the number of lymphocytes takes place. On the other hand, in those cases of tuberculosis which are recovering the lymphatic apparatus also regains its functional activity and lymphocytosis is again in evidence. Indeed, it may be assumed that lymphocytosis in tuberculous patients is an evidence of improvement or recovery, and it has been further suggested that in tuberculosis patients with high lymphocyte counts give the best prognosis.

In syphilis there may be considerable increase in the number of lymphocytes when there is lymphadenitis. This is observed

in the second stage of acquired lues and is sometimes observed in the congenital form of the disease where enlarged lymph-glands occur. Mononucleosis may sometimes be observed in congenital lues with lymphadenitis.

Lymphocytosis may occur during convalescence in many of these acute infectious diseases. It has been shown to occur after vaccination. It has already been pointed out that it occurs in typhoid fever, malaria, influenza, varicella, and variola. After the eighth day of a moderately severe scarlet fever the lymphocytes may constitute 50 to 60 per cent. of the total number of white cells.

Most of the acute infectious diseases, mild as well as severe, may show a post-infectious lymphocytosis which may last for weeks and months.

In certain protozoic infections, trypanosomiasis and filariasis, an increase in the number of lymphocytes has been observed, and this is also true after infestation of the intestinal tract with worms.

One approaches the discussion of the relation of lymphocytosis to the endocrine glands with caution because the observations are not uniform and the opinions are diverse.

Kocher thought that lymphocytosis varied directly with thyrotoxicosis and was an index of the severity of the toxicity of the thyroid secretion. This observation has not been confirmed. Severe cases of Basedow's disease may occur without lymphocytosis, and, on the other hand, there may be a decided increase in lymphocytes in hypothyroidism or myxedema.

The same blood reaction has been shown to be present in disturbances of other ductless glands. C. J. Klose thought that hyperplasia of the thymus gland may produce mononucleosis, though the subject of the thymus gland is as much mixed in this connection as it is in general. Some authors have maintained that if the thymus gland was removed decrease in the leukocyte count was observed, and by the injection of thymus extract lymphocytosis was produced. Other authors could not confirm these results. They found hyperplasia of the thymus without lymphocytosis, and, on the other hand, observed marked

constitutional lymphocytosis without a trace of thymus hyperplasia. It seems to be true that lymphocytosis following injection of thymus extract is not a specific reaction.

On the whole, it must be said that the cause and significance of lymphocytosis has not been satisfactorily answered or fully established. Some investigators seem to draw far-reaching conclusions which are not justified by the known facts. This statement seems to apply particularly to the relationship of lymphocytic increase to the glands of internal secretion.

The relation of the lymphocytes in the blood to the action of the autonomic nervous system has received wide-spread attention and has been the subject of an interesting discussion. This subject was opened particularly by the studies of Eppinger and Hess, in their discussion of vagotonia. They assume that this condition was associated with a characteristic eosinophilia. They thought that injection of pilocarpin produced an eosinophilia, while injection with atropin and adrenalin caused the eosinophils to disappear. Whether these observations were correct or not is immaterial at this point. Publications by Eppinger and Hess opened the question whether variations in the blood-picture could be due to a neurogenous cause. The observations of Eppinger and Hess concerning eosinophilia failed of corroboration by numerous investigators. Nevertheless, it was suggested that certain glandular disturbances might be produced by changed conditions in the autonomic nervous system.

Dysfunction of the autonomic nervous system might be produced in a variety of ways. In acute infectious processes toxic products might be assumed to act directly on nerves or ganglia, or mechanical or chemical injury might similarly affect these nervous structures, thus secondarily affecting the growth, nutrition, or functional activity of lymph-gland structures.

Injection of adrenalin may temporarily cause a definite increase in the lymphocytes, associated with an increase in the total number of leukocytes, though after a short interval the number of lymphocytes becomes decreased. The same is true if pilocarpin be injected: first there is an increase in leukocytes, later a decrease. Indeed, experimental lymphocytosis may be

produced by the injection of vasoconstrictor substances, such as adrenalin, muscarin, pilocarpin, and barium chlorid. This is probably a mechanical reaction produced by contraction of the arterioles of the spleen.

Lymphocytosis is not uncommonly associated with anaphylactic reactions. It is difficult to say whether this is a specific protein reaction of the blood or whether it is simply a post-toxic lymphocytosis.

An increase in the number of lymphocytes may be produced after therapeutic injections of iodin and its derivatives, phosphorus, arsenic, tuberculin, and thyroid extract. It also occurs occasionally after the ingestion of alcohol. The number of lymphocytes may also be increased after treatment with radium and *x*-ray, probably produced by lymph-gland stimulation.

In this brief review it has been pointed out that the number of lymphocytes may vary considerably during health, may be influenced by age, climate, diet, activity, and exercise, and also that a number of infectious diseases may cause a relative increase in the lymphocytic elements either during the height of the disease or during convalescence. A number of facts have been assembled which should assist the reader in interpreting the variations in the lymphocytic content of the blood as well as to point out the occasional difficulty of differentiating a high lymphocyte count from a grave and fatal blood disorder.

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### PHOBIAS AND NEUROLOGY OF THE VISCERA

WHEN one stops to consider to what degree the pathology of the peripheral nervous system has been developed it is striking that the pathology of the nervous system of the viscera is practically unknown. One of the main reasons for this has been the scanty knowledge of the physiology of the visceral nervous system. Another, is the refusal of physicians to admit the possibility of neuropathic origin for many visceral disturbances; for example, mucous colitis. Too often the conception of the etiologic factor has been deduced from reasoning which is doubtfully logical. If, prior to the development of a mucous colitis, constipation was present and had been injudiciously managed by promiscuous catharsis, then this was assumed to be the cause of the mucous colitis. The psychic factors underlying the causes of the constipation, and particularly the urgency for exhaustive catharsis, and the peculiar and bizarre dietary habits and chronicity of the disorder are often overlooked.

The relation between psychic function, for example, emotion, and physical reactions is generally recognized. One is no longer mystified at the existence of symptoms when no structural disease exists. If one is frightened the hair stands on end, the mouth becomes dry, the heart beats rapidly, sweating is present, shivers run up the back, the face is pale, the pupils dilated, secretion of the stomach ceases, involuntary urination may occur, and diarrhea is common. Certainly if an individual were in a chronic state of fear he could be accounted as a sick man, suffering from very distressing symptoms. The symptoms which occur in conjunction with emotion are logically the result of nervous activity, stimulation of certain parts by some and inhibition of

other parts by other nerves. It is not strange, therefore, that we may have some of these symptoms reproduced by stimuli other than emotion; for example, drugs and disease. Toxic goiter produces sweating, tachycardia, and diarrhea; injection of adrenalin may produce a combination of physical symptoms, tachycardia, shivering, apprehension, cardiac oppression, leading to a condition of indefinable great fear. Pilocarpin produces urgency in micturition and defecation, sweating, diarrhea, and at the height of action of pilocarpin animals evacuate not only large amounts of feces, but also characteristically glairy tenacious mucus which is as coherent as a band.

The viscera are innervated by the vegetative nervous system. This may be divided into two parts: One, the sympathetic, is represented by the thoracolumbar portion of the sympathetic system which goes to make up the splanchnic, the cervical, and the sacral sympathetics. This portion is composed of non-medullated fibers originating in the cells of the lateral horns of the spinal cord; the other, the parasympathetic system, consists of medullated fibers emerging from the gray matter of the midbrain and medulla (cranial outflow), and the sacral region of the spinal cord (sacral outflow). The bulbar portion originates in the nucleus plexus of the glossopharyngeal-vagus nerve, and participates in the innervation of the visceral content of the thorax and abdomen. The sacral portion originates in the cells of the anterior horn of the sacral part of the cord and emerges as the pelvic nerve.

Two distinct systems, therefore, are included in the vegetative nervous system. They are mutually antagonistic, the thoracolumbar portion being opposed by the sacral parasympathetic. In addition to these two systems there exists an intrinsic nervous mechanism consisting of a network of ganglion cells and fibers situated within and on the walls of the viscera which plays an important part in the regulation of the various automatic motor activities of the gastro-intestinal tract.

The parasympathetic system is the motor mechanism for the longitudinal and circular layers of the intestine, while stimulation of the sympathetic produces, according to some, a general

increase of tone, and, according to others, a contraction only of the sphincters. The parasympathetic system initiates digestive secretions, whereas the sympathetic inhibits them. We can express the motor disturbances of the alimentary tract under the heads of irregular and exaggerated contractions, tonic spasms, and atony. Irregular and exaggerated contractions are due to irritation of the parasympathetic and when in the vagal area produce colic; when in the pelvic area, tenesmus. Tonic spasm and atony are both due to sympathetic irritation, spasm due to a contraction of the sphincters, atony due to inhibition of normal movements.

Some years ago Eppinger and Hess attempted to establish a specific symptomatology for the two divisions of the vegetative nervous system. Largely by the use of pharmacologic methods they separated the complicated symptomatology of these systems into two groups; one, of parasympathetic origin, was termed "vagatonia," the other was called "sympathicotonia." Broadly speaking, both syndromes were characterized by hyperexcitability of their respective systems. As pilocarpin stimulates the parasympathetic, symptoms of vagatonia could be elicited or aggravated by its administration. Adrenalin, on the other hand, would aggravate an already existing sympathicotonia. Included in the symptomatology of vagatonia was spastic constipation and diarrhea from increased secretion and increased peristalsis. The diarrheas were easily explained upon the basis of vagatonia, since it is known that pilocarpin will cause this condition in an animal. As a matter of fact, it is associated with the vagotonic symptoms of Addison's disease and Graves' disease.

Although their work was based upon anatomic, physiologic, and pharmacologic data, it has by no means attained general acceptance and certainly is only partly true. Many of the symptoms attributed to vagatonia are difficult to explain upon the basis of stimulation of the vagus; for example, the constipation. However controversial the theories of the selective action of these two systems may be, the fact remains that stimulation of them does produce modification of function of the motor and secre-

tory elements of the bowel. That these systems can be stimulated by psychic influences need not be argued further.

Instinctive and reflex acts may be modified by experience. If an animal with a salivary fistula has presented before it a dish of food, there is an increase in the flow of saliva; if an animal with a gastric fistula is shown food there is a flow of gastric juice. If now the animal is annoyed, the salivary and gastric secretions cease. A similar phenomenon appears in connection with the movements of the intestines. If simultaneous with the showing of food a bell is rung and the animal is permitted to eat the food and the experiment is repeated a number of times, a time will come when ringing the bell alone will produce a secretion of saliva and gastric juices. This is a conditioned reflex. We can see, therefore, that experience may condition a reflex so that it can be brought about by a stimulus which under ordinary conditions is quite indifferent and that the normal response to a specific stimulus may be inhibited. This mechanism plays a very important part in the formation of certain habits, types of behavior, and function which may produce various disturbances of the function of the colon.

Phobias, or fears, relating to the intestines are a common occurrence. A man forty years old presented himself for consultation with the complaint that he had never been able to ask a young lady to accompany him to the theater or any social function, and as a result, despite his yearning for a loving wife, he was forced to remain a bachelor. When a boy he was taken to a famous and glittering restaurant and there, because of his excitement, he soiled his trousers. Ever since he had been fearful of a repetition of this accident, and as time elapsed he was forced to go to the toilet more and more frequently, before he partook of breakfast, after breakfast, before he went to work, several times on the way to the city, and so frequently during the day that no sustained effort of work or pleasure was possible. It was impossible for him to travel on express elevated trains, to go to church, or the theater, to social functions, or, in fact, to occupy any profitable part of society. When a full explanation was offered to him that the integrity of function of defecation

was dependent upon a normal nervous system, that the examination proved that he possessed such a normal system, and that it was an impossibility for a repetition of the distressing circumstance to recur, he was able in a very short time to so rapidly diminish the number of his enforced evacuations that he rushed into the office one day exclaiming that he was engaged—thereby proving the efficacy of the cure.

Some of these patients have formed stools, some in whom the condition began with an attack of organic diarrhea may, upon the persistence of efforts at evacuation, have quite unformed stools which, when psychotherapy is employed and the interval between visits to the toilet lengthened, become normally formed.

Constipation phobias are even more common. They occur most often in individuals who have been warned against the evils of constipation, perhaps when suffering with high blood-pressure, nephritis, hemorrhoids, etc., and who, undirected, exaggerate the medical advice in a most phantastic way. Usually they are not constipated at all, but because of their introspection have developed visceral sensations which are interpreted as necessity for emptying the bowel. Rarely do they feel satisfied that the bowel is completely empty and they may remain seated upon the toilet for hours.

Diarrhea due to emotional disturbance is very common and I suppose that in the memories of our own experiences we will find that upon some occasion, as when engaged in writing an examination, taking a final quiz, addressing a medical society, or confronted with danger—as in warfare, that a diarrhea occurred. There are many classical examples of men and women who have been forced to relinquish their public work or pleasure because when attempting to address an audience or go to an exciting social function a diarrhea regularly developed. Such a simple disturbance of the intestinal function may become the groundwork for the development of a severe psychogenic disease of the bowel.

Nearly all the neuropathic constipations are due to a conditioned reflex. They are the constipations due to education. They may occur upon the basis of painful defecation due to

hemorrhoids, etc., to bad habits or irregular or delayed visits to the toilet because of inconvenience, embarrassment, seeming lack of time or opportunity, and laziness. Often they are produced by the establishment of a habit of having a movement only by artificial means, as suppositories and enemata, without any attempt to have a normal passage. Dejerine once saw a man who had taken 15,000 enemas in his lifetime. I purposely omit here the use of cathartics, as they produce a mechanism, which although primarily based on a neurogenic factor, introduces another element which eventually produces secondary disturbances of the colon.

One of the choice controversial subjects for discussion along with surgical or medical treatment for gastric ulcer is mucous colitis. In 1913 Dejerine stated that "here is a disease which thirty years ago was almost unknown or at least seen so little that one could easily count the cases. Now in these later years it has become so wide-spread that in many watering places as well as in a considerable number of sanatoria they treat this trouble almost exclusively. Characterized essentially by glairy and mucous stools, sometimes accompanied by false membranes, by alternate attacks of diarrhea and constipation, by painful sensations in the region of the large intestine, just how can one tell whether or not the symptoms which constitute a mucous colitis are likely to be nervous in their origin?" We have seen that constipation and diarrhea alike may result from psychogenic causes. Whether mucous hypersecretion of the intestines is a defense against a neuropathic constipation enhanced by continued use of cathartics, or is the result of stimulation of the parasympathetic system, as is suggested by the action of pilocarpin, seems to me to be of academic interest only. That the disease may be controlled by proper diet and habits and removal of cathartics is in no way a proof of its cause or pathogenesis. The important factor is the determination of the underlying cause for either the constipation, the diarrhea, or both. Certainly the condition is not the result of any aggregate neuropathic conditions. Neurasthenia, anxiety neurosis, conversion hysteria, hypochondriasis, and so on do not necessarily produce it. It

may, however, result as a specific reaction in any of these conditions, and may depend upon various patterns of behavior.

A young woman, a member of a well-bred family, suffered the loss of an eye early in her childhood. She was conditioned to a type of behavior characterized by shyness, sensitiveness, embarrassment, and self-consciousness. She was unpopular and unsought for, spent much of her time in romancing and dreaming, obtained a fair education, and entered adult life. She was excluded from normal social contacts and was confronted with the necessity of earning her livelihood. Unconsciously seeking tenderness and attention, she took refuge in an illness which was precipitated by an acute gastrointestinal disturbance. She rapidly developed the picture typical of mucous colitis. She became a burden on her family, an unhappy, emaciated, feeble, miserable creature suffering from cramps, pains, and diarrhea. Her mother and brother were forced to carefully guard her and care for her, to nurse her, and to be tender with her. Every slight pain produced a paroxysm of whimpering fear, each attempt at occupation, and typically, it was teaching defective children, working in a library or an art store, produced an exaggeration of the symptoms, preventing further occupation. Diet after diet was prescribed and a tender sirloin steak would have been as quickly eaten as an arsenical paste. Yet this patient, isolated in a hospital from her friends and relatives, prohibited from writing or receiving letters, from reading fiction, and forced to eat liberally of a general diet, did not, to her surprise, die, but was able to digest and assimilate her food and gain weight. Of course we cannot give her another eye, but she may be taught that there is as wide a visual field to one as to two eyes, if she but moves her head a bit.

Now it does not follow that all cases diagnosed as mucous colitis are neurogenic in origin. It is exceedingly likely that this name covers a multitude of diseases, some of which will be found to have one and others another cause. It is true, however, that a great number occur in neuropathic individuals and that unless one discovers the particular maladjusted conflict which is the cause of the production of the underlying soil a permanent cure will not be effected. It is of the greatest importance to recognize the neuropathic case, inasmuch as undue insistence upon a particular diet may only serve to fix the phobia of eating as it does in the neuropathic gastric disturbances. In both conditions the patients develop a habit of eating dependent upon their conception of what is best tolerated. Perhaps at first they avoid things which are hard to chew or difficult to swallow. Quantity appears then as a factor; given small quantities of a certain

type of diet they take it readily, given a single large quantity the psychic impression of difficulty of eating or digesting produces a fear that the food will disagree with the patient. In the end, these patients get to the point where they can take but soft food, semiliquid or liquid, and only in small quantities. So bizarre are the self-imposed diets that upon one occasion I saw a patient who was convinced that unless she adhered absolutely to a diet of prunes and lettuce death was inevitable.

Parts of the gastro-intestinal tract other than the colon are as frequently affected by psychogenic causes.

Anorexia, although often appearing in the course of hysteria, is also seen in relation to phobias, and is usually associated with the same mental mechanism as that producing disturbances in the colon as the result of very assiduous and bizarre attention to the diet. At times appetite may be present in the normal way, but is rapidly inhibited by a fear, as in the following case:

A man aged twenty-six presented himself for examination because he had for a number of years been unable to finish any meal. This he explained upon the following basis: He would be able to sit down to a meal, whether at home or in a public restaurant, but as the food would be brought to him, he would experience after a few mouthfuls a sense of fulness and distress about his heart, followed by marked palpitation and air hunger, and he would be forced to immediately pay his check and leave the dining table. Careful examination showed no evidence of any organic disease, but further inquiry into the history of the case brought out the following facts: He was able to fix the exact date for the beginning of his disability to a time when, seated at a table, the above mentioned train of symptoms occurred and he was forced to rush out. When questioned as to the exact nature of the feeling he denied the existence of any fear. It was, however, explained to him that the symptoms were quite compatible with a fear reaction, that no organic disease existed, and that nothing could happen to him despite any feelings he might possess. He immediately arose from his chair, became rather highly excited, called me an old fool, and rushed from the office. Several days later he returned, and upon entering the office stated that first he wished to apologize for his unseemly behavior, and, second, to fully agree with what I had, perhaps bluntly, stated to him.

He said the reason he was so greatly disturbed was that he had always considered himself possessed of unusual courage and the contemplation of any yellow streak was very obnoxious. During the war he had been an aviator, was credited with a

number of victories, and had been decorated by the government. With this background the accusation of possessing fear seemed somewhat incongruous. Thinking the matter over calmly, however, he recalled that a number of years before on passing a barber shop he observed a commotion therein, and entering found his favorite barber lying on the floor having a fit. He was considerably disturbed by this and thought it would be a terrible thing to have such an occurrence in a public place. He was now able to identify the feeling he had upon that occasion with the feeling he experienced in the restaurant upon the occasion of his first attack.

Not only do fears but other well-recognized emotional states moderate the degree of appetite, salivary, and gastric secretions, as was indicated formerly in relation to the explanation of colonic disturbances. Grief is a well-recognized cause of anorexia, and the hearty partaking of a dinner is incompatible with the expectation of death.

Vomiting, likewise frequently found in hysteria, may, because of a mechanism of fear occur in other neurotic states. The sensation of fulness after eating, giving rise to fear of gastric disease or pressure upon the heart, conditioned as it is by the pernicious type of advertisements of patent medicine, calls the attention of the patient to this particular function and brings forth an effort to relieve himself of the discomfort, which is followed by an attempt to retch, often assisted by stimulation of the vomiting reflex by inserting the finger into the throat, to the end that vomiting follows regularly upon the ingestion of food.

Just as we have observed a fear of incontinence of feces, so frequently do we see a fear of incontinence of urine. Particularly do we meet with this condition in women, and a very marked frequency of urination during the day in the absence of any pathologic findings in the urine, should call our attention to the possibility of a psychogenic cause.

The false cardiopaths are among the most frequent of the cases conditioned by fear. A man having injudiciously partaken of a large banquet, seated in a street car reading a news-

paper, is aware of a sense of discomfort somewhere about the left upper quadrant of his abdomen. Coincidentally he happens upon an article describing heart disease, and the thought occurs to him that perhaps he has some trouble with his heart. At this moment something has occurred to the man, namely, that his attention is directed to his heart. Upon reading this the reader will be conscious of the position of his right hand, whereas a few moments ago such a sensation was absent from his consciousness. The slightest stimulation with a wisp of cotton is immediately appreciated by our skin, yet none of us is conscious of the tremendous pressure of our bodies against the skin of our buttocks until our attention is directed to it. One has only to remember the method of torturing during the days of the Inquisition, wherein a drop of water was permitted to fall at regular intervals upon the scalp to appreciate the great sensitiveness which may be developed by attention. Our patient decides to consult a physician, and upon doing so is told that he has nothing the matter with his heart, and that he shall "forget it." Inasmuch as he is conscious of some sensation which he did not formerly have, it is difficult for him to forget it, and so he consults a specialist, who, after a more time-consuming examination, assures him that there is nothing wrong with his heart and that he shall "forget it." The patient now decides that either the physicians are withholding their real opinion to avoid frightening him or that he is suffering with a disease so subtle that physicians are unfamiliar with the condition, and begins to defend himself against any dire result.

Whereas before he was able to run to catch a street car he now barely walks. Fearful of losing consciousness, even of dropping in the street, he no longer is able to go out alone. He does not visit places of amusement and if he is forced to go to the theater he always occupies a rear seat so that if any peculiar sensations occur, and they regularly do, a sense of insufficient air, palpitation, a feeling of fulness, he will be able quickly to escape into the open air. He is unable to ride on an express elevated train because he cannot get off immediately upon experiencing any peculiar sensation. He is no longer able to take

journeys on a train or on boats. He gradually and progressively becomes more and more separated from society, and is finally in as pitiful a condition as any one suffering from the most serious organic disease.

The part of the body to which fear is attached is largely a matter of accident. The mechanism is exactly the same. If a man develops a chronic headache, let us suppose an indurative or rheumatic headache, which stubbornly resists our best efforts at treatment, and accidentally hears of a friend of his who has lost his mind and remembers that his friend had suffered with headaches, a fear of insanity may very likely develop under proper conditions. Such a man will complain of terrific and continuous headache, always increased by attention concentration, and mental activity. The pain may begin with a slight, but real pain, and now be transformed to a feeling of fulness, of head pressure, of "water trickling in the head," of "snapping" in the head. When we ask such a patient, "Suppose you had the pain in the foot, would it bother you as much?" The answer invariably is, "Of course not, I have often had worse pains in my feet, but this is at the base of the brain." It is, therefore, not the pain which produces the disability, but the fear of the condition that it may denote. In this particular instance fear of losing his mind causes the patient to examine himself, usually by attempting to memorize, and because of his divided attention between his apprehension that he will not memorize successfully, and the attempt to memorize, his impressibility is poor, and therefore he is actually unable to successfully memorize. He immediately draws the conclusion that he has already lost his mental faculties. This, in turn, gives rise to additional fears, producing further symptoms. He is now unable to read the newspaper, because if he were to read of any one who has become insane or committed a crime or outrage, or suicide, he is fearful that the emotional disturbance would be likely to cause him to commit the same offense. He begins to avoid excitement, he demands perfect quiet in the home, and within a short time excludes from his experience as many mental stimuli as the cardiopath does physical stimuli.

Much the same mechanism is present in the fears of paralysis, or a stroke, or syphilis.

Not only are fears encountered in the milder types of mental disturbance as the neuroses, but frequently we encounter them in the course of a more serious neurosis or psychosis. Melancholia and the depressed phase of manic-depressive insanity frequently exhibit many fears which do not differ from those encountered in the neuroses, with the exception of their marked fixity and their inaccessibility to persuasion and explanation. These fears are, of course, compatible with the unhappy mental viewpoint of the patient and consist largely in fears of death, of incurable illness, chronic invalidism, and insanity.

Occasionally one encounters bizarre fears incompatible with any reason, as for example, the following:

A young woman, aged twenty, appeared for consultation, and it was observed that her hands were macerated and excoriated. The history revealed that she was continuously washing her hands, not only with soap and water, but with various types of cleanser, such as are used to clean sinks and toilets. Some time before she had read in the newspapers that, owing to the Eighteenth Amendment, bootleggers were using wood alcohol to cut whisky, and that as the result a number of deaths had occurred. Thinking about this she wondered if it would not be possible to carry such a poison and by contact with another person produce his death. This quickly resolved itself into a fear that she might be contaminated with wood alcohol and touching her father be the cause of his death. As a result she refused to go near her father and began to protect herself against the possibility of contamination by avoiding all places where wood alcohol might be used. So thorough was her investigation of the distribution of wood alcohol that it was almost impossible for her to remain even in her own room, because of course, druggists deal in it, paint shops use it, it is a common diluent of dyes, many artisans employ it, so that she was forced to wash her hands following contact with persons or any objects. Fear that the laundress might come in contact with this substance caused her to begin washing her own clothes, and she could not place them in a bureau drawer because of the dye and paint on this article of furniture. This continued to such a degree that her entire waking state was occupied with continuous washing and rewashing of her hands, clothing, and bedclothes, the washing liberally assisted by copious tears.

The very nature of this fear called our attention to the possibility of the existence of a more severe type of nervous dis-

turbance, and all psychotherapeutic measures failed to modify this fear. Further observation of this patient revealed the fact that she was suffering with dementia praecox.

The fear of harming some one is very common and leads to an attempt to escape by avoiding such dangerous objects as scissors, knives, and razors. At times this attempt to escape assumes ridiculous ends, such as the avoidance of tacks, pins, and hairpins. It is possible for such a fear to attain an absurd degree and still be part of a neurosis, as was the case in a patient who, following a rather mild attack of influenza, awakened one night from a frightful dream in a somewhat confused state and remained frightened for the several minutes required for her to fully awake. She then developed a fear that she was losing her mind, and that she might at any time lose control of herself and injure her mother. As a result, she avoided all sharp objects, even requiring her mother to stop wearing hairpins. Subsequent observation of this patient proved that she was suffering with an anxiety neurosis.

Obsessions and compulsive ideas are often really based upon a fear. For example, a young society matron who occupied a box at the opera every week during the entire season, has never been able to completely hear a single opera during her life. From the moment that she enters the auditorium she is compelled to count all of the electric light globes, and unless this count comes out in a multiple of three she must recount them. Considering the large number of these fixtures in so large a hall, it can readily be seen that her attention would probably be occupied quite thoroughly throughout the duration of the opera. This compulsive idea is based upon a fear that unless she is able to relieve the sense of uneasiness or oppression which is present before the lights are counted and come out in a multiple of three, she will lose control of herself and create a scene.

Such ideas are, of course, closely related to ordinary superstitions and fetishes of luck. You all may remember that as children we would never step on a crack in a sidewalk, or pass a paling fence without touching every seventh paling lest we be followed by bad luck. The mechanism of the more complicated

obsessions and compulsive ideas is much the same. We fear only that which is unfamiliar to us, and the hereafter is feared by us all. It is logical, therefore, that most of the fears which we encounter are those dealing with such conditions concerning which the patient has no reliable information, and although his observations may be accurate, his interpretation is always wrong.

It is, of course, not sufficient to discharge such a patient suffering with any fear with the bald statement that he is physically well and that he should forget it. Were it possible for him to forget it, he would never consult a physician. Although the psychogenesis of these fears remains a somewhat controversial subject, and the discussions upon pathologic psychology dealing with fears and obsessions may lead one into distant realms smack-ing of metaphysics, if the physician is able to give relief to a patient neither he nor the patient much cares what the underlying mechanisms may be. Not every one is a potential phobic. These fears occur in certain individuals who are more impressionable, suggestible, and who are unable to properly or successfully compensate for the things in life which they are denied, or comfortably do the things which are required of them. Whereas an average person may be able to successfully store the vicissitudes of life and philosophically accept numerous defeats, some have produced a mechanism, perhaps reflex, which is intended to shield them from assuming their responsibilities. It is well known that certain fishes when touched assume an absolutely immobile position produced by the death-shamming reflex. All of us are familiar with the behavior of the opossum in this respect. So not only in man but in the lower species as well are found types of behavior which have a protective function.

It is not always necessary, however, although it is very fruitful, to determine the underlying conflicts which condition a neurosis. Although it has been stated, probably correctly, that the removal of a symptom is not the cure of a disease, yet it would be impossible to bring every patient suffering with a neurosis characterized by fear to a psychiatrist or to a psych-analyst who, by virtue of the great time required for analysis,

could see but a very limited number of patients. Any physician who is experienced and who has the confidence of his patients has always practised that type of psychiatry which is concerned with the adjustment of the conditions of life, and the physician as often as the priest has been the repository of confidences which have relieved and cleansed the patient of headaches and troubles.

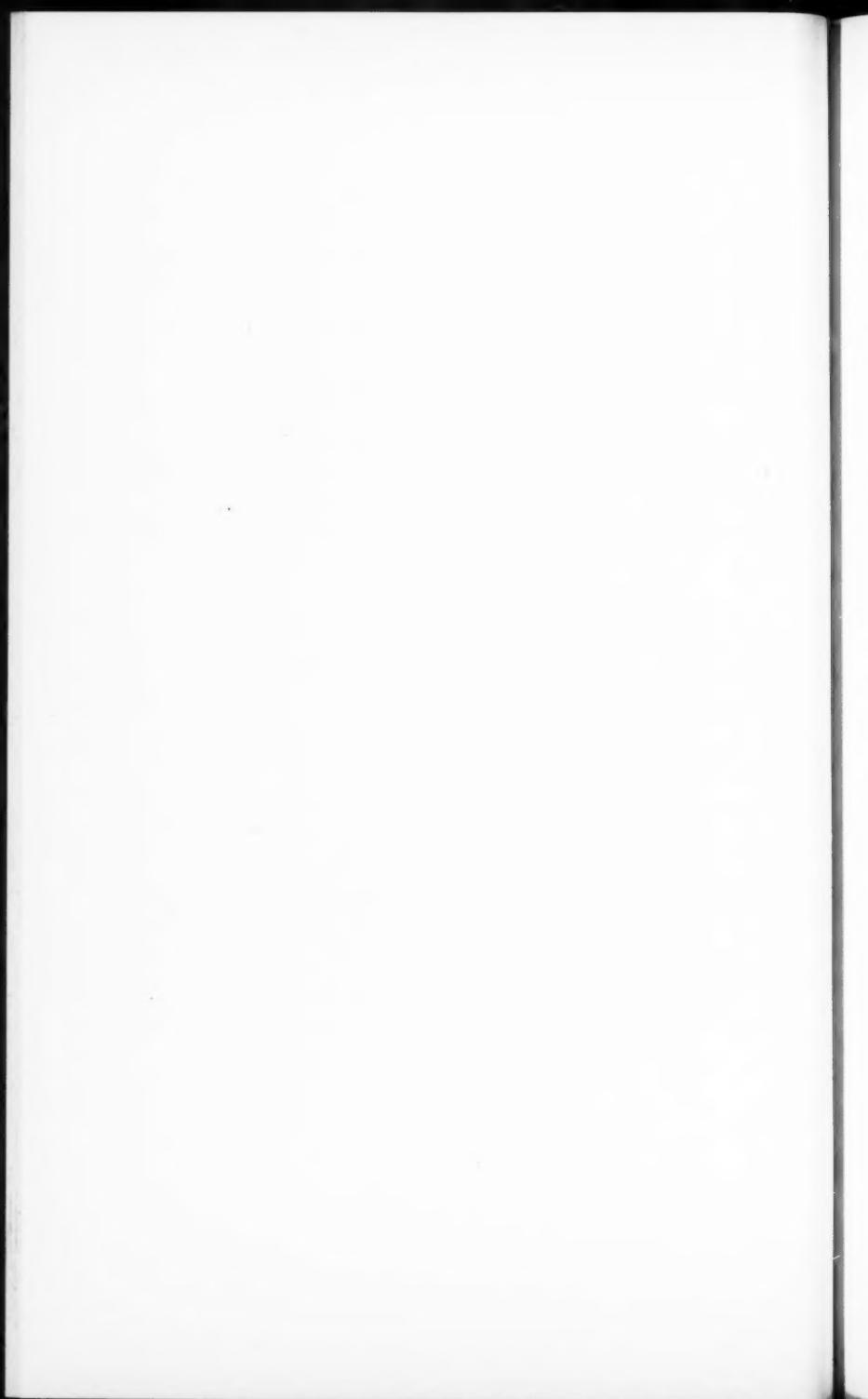
The removal of the symptom is, fortunately, far more simple than the reorganization of a patient's character. It would be foolish and futile to treat a man who believed that the earth was flat by the administration of elixirs, pills, massage, and electricity. Such a condition would be treated by the presentation of fact, by an astronomer and a mathematician, to the end that the man would modify his idea correctly. The same mechanism is employed in the treatment of fears. The first requisite is faith. If a child is afraid of a dark room it must first be instructed by some one whom it trusts, as the mother, that there is nothing in the dark room that does not exist in the light room, and if the child believes in the mother and knows that she would not expose it to any danger it is ready for the next step. This consists of gradually facing the fear. It would be cruel and illogical to thrust this child unceremoniously into a dark room and shut the door. This could only result in increasing the fear and producing a panic, but the child, having been instructed by one whom it trusts, may be allowed to remain in the dark room with the door fully open, the mother talking to the child outside, and day by day the door may be gradually closed until the child remains comfortably in the darkened room. This is applicable to any fear. If a man fears that he will lose consciousness, drop dead, create a scene, or have a fit in a restaurant, here, as before, the first requisite is faith. He must be very carefully and completely examined, and if no organic disease be found he must be so instructed. This, however, is not enough, because he suffers with sensations the meaning of which is a mystery to him. He must be shown that such sensations can be the result of emotional disturbances. Just as blushing occurs from hearing a naughty story, so it may be demonstrated that other changes in other

tissues of the body occur, as sweating, trembling, tachycardia, precordial oppression, heat sensations, etc., etc., likewise can occur in the absence of organic disease. The patient must be made to realize that the sensations he complains of are real, not imagined, not hypothesized, but that they are common sensations, that they are all due to emotional changes, and that no matter how he feels nothing can happen to him.

The next logical step is to induce him to face those experiences of which he is fearful. If he is afraid to walk alone, the first day he may be accompanied in one direction for two blocks and instructed to walk back alone. Daily this is increased until finally within a short time he is ordered to walk alone; then to ride on street cars, then to enter into all the activities that a normal individual would encounter, despite his feelings. Being fearful of reading newspapers, he is commanded to read the first column on the first day, the first two columns on the second, and so on until he is able to read the entire newspaper. Continuously he must be assured that nothing can happen to him, irrespective of the feelings which he experiences. At this time the patient will always tell you that he has already made himself do these things, but a very homely illustration will suffice to show that his mechanism is not the same as yours. If a man were to come into one's office and point a gun at him, threatening to shoot him, one could not continue his normal occupation in comfort. When a well-meaning friend would remark that he believed the man was only bluffing, it would be difficult to accept this with assurance. If, on the other hand, some expert were to appear and would say, "You need not fear, because the gun is not loaded," one could continue his occupation in perfect comfort. The gun represents the symptoms, and although one might not know when the man would shoot, but forces himself to continue his occupation, he would remain fearful until he knew that the gun was not loaded.

Although such simple explanations suffice to dispel these distressing fears, one is astonished at the large number of individuals who have never had their fears explained to them. It is obvious that it is necessary, first, to recognize the existence

of real sensations that are not dependent upon organic disease. Second, that such sensations are as amenable to treatment as are organic diseases. Third, that they should be treated, and that the exercise of ordinary homely horse sense and advice frequently suffices.



## CLINIC OF DRs. JULIUS H. HESS AND S. L. BERMAN

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### ERYTHEMA NODOSUM IN CHILDHOOD

THE pediatric literature from European clinics strongly stresses the intimate relationship of erythema nodosum with tuberculosis. (Observations by clinicians from other countries suggest the lesser frequency of a tuberculous etiology in this condition.)

In the American literature infections related to the streptococcus group have, until more recently, been given first place as causative factors, with tuberculosis secondary in importance. Other conditions with which it has been associated are syphilis, rheumatism and its complications, measles, diphtheria, malaria, and the ingestion of drugs, more especially the iodid and coal-tar group. It has also been considered as a specific disease. According to Rosenow, erythema nodosum is caused by a diphtheroid organism, having a special affinity for the subcutaneous tissues.

During the past ten months 3 cases have been observed at the University of Illinois Research Hospital in which the clinical, roentgenologic, and tuberculin reactions point to tuberculosis as an underlying etiologic factor.

#### CASES, TUBERCULIN POSITIVE

**Case I.**—Edward H., white boy, age three years, ten months. Admitted 3/16/27.

*History at Entrance.*—Child was well, played vigorously until six days before entrance, when a large number of reddish spots appeared on his legs. These spots were raised, very tender. Movement caused pain so that the child had to be carried. No history of trauma. No other complaints except cough for two days previous.

Physical examination was essentially negative except for somewhat enlarged and slightly inflamed tonsils, and enlarged anterior and posterior

cervical glands. Chest showed diminished resonance over right intrascapular region. No definite auscultatory change.

Over the anterior surface of the lower extremities, extending from the knees to the ankles, there were many areas, red in color, with a violet tint in it. These areas were from about 1.5 to about 2.5 cm. in diameter, somewhat oval in shape, raised, poorly circumscribed, warm to the touch and very tender. Any stretching of the skin of the legs, such as would occur with movement, causes the child to cry with pain.

3/16: Pirquet done on entrance was strongly positive.

3/25: Eruption gradually faded. Swelling and tenderness disappeared. Gone by 3/25.

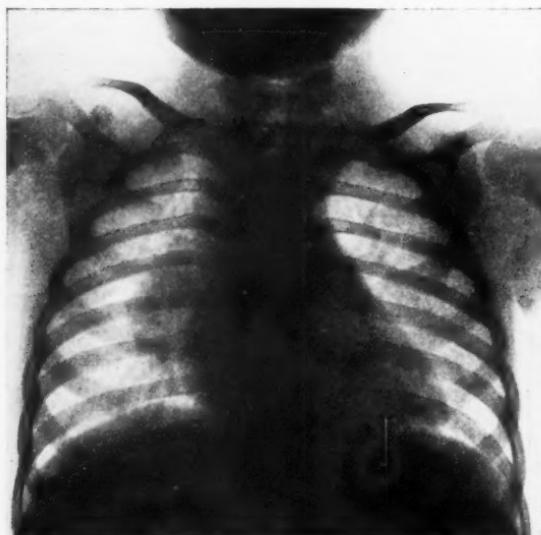


Fig. 3.—Case I. Tuberculous erythema nodosum.

3/25: Cantharides plaster applied. (Kundratitz<sup>9</sup> found that the serum obtained from cantharides blisters on erythema nodosum patients activates tuberculin and that the Mantoux reaction obtained with tuberculin diluted with this blister fluid is stronger than that resulting from an equal dilution of tuberculin and sodium chlorid solution.) In this case we obtained equal reactions with both solutions.

3/29: 1/10 mg. O. T. injected intradermally.

3/29: x-Ray plate shows a triangular hilus shadow on the right side with base toward mediastinum and apex toward periphery. An interlobar line is also present extending from apex of this triangle.

3/30: At the seat of inoculation there appeared a markedly indurated

vesiculated area, surrounded by an erythematous zone. Indurated area 2 x 2.6 cm. Erythematous zone 4.4 x 3.6 cm.

4/16: 1/100 mg. of tuberculin was injected subcutaneously. (Ernberg<sup>8</sup> obtained in this manner a recurrence of the erythema nodosum lesions in 5 out of 11 cases.) Our results were negative. Up to this time his temperature had ranged between 97.8° and 100.2° F. Following the subcutaneous injection of tuberculin, twenty-one hours later his temperature dropped to 97° F., and the following day rose to 103° F.



Fig. 4.—Case I. Note lesions on legs.

4/18: There was a marked local reaction at the site of injection. At this point an indurated area developed which was 5 cm. in diameter, surrounded by an erythematous zone 8 x 12 cm.

4/19: There remained only an indurated area 3.5 cm. in diameter.

4/22: He developed measles and was discharged to the Contagious Hospital. While at the hospital he further developed scarlet fever and later diphtheria. He remained at the contagious hospital for about four months.

9/9: A social service nurse visited his home and reported him as looking well and going about in his bare feet.

**Case II.**—Robert R., white boy, age five and a half years. Admitted 9/29/27.

*History of Exposure.*—He was exposed to his grandmother, who died of tuberculosis three weeks before he entered the hospital. He was in close contact with her from July 1 to September 1, 1927.

Child was well until two weeks before, when he began to show general malaise, with loss of weight. About six days before entrance red nodules appeared on his legs and arms. For the past four or five days he has complained of pain in legs of an aching character. Fever for past few days up to 105° F. Heavy sweats during this time.

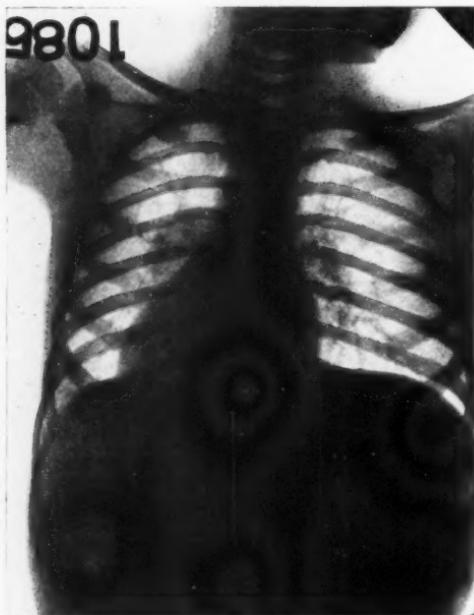


Fig. 5.—Case II. Tuberculous erythema nodosum.

Past history essentially negative.

*Physical Examination.*—Essential finding is that of numerous raised, dusky red (bronze-like) glazed, tightly stretched, very tender areas on the anterior surface of legs and thighs and one above left elbow. Nodules are 0.5 to 2 cm. in diameter.

There was a general adenopathy, with glands of small size.

Chest shows slightly diminished resonance over right base posteriorly with distant breath-sounds and increased vocal resonance. No râles.

Mantoux 0.1 mg., markedly positive in eighteen hours.

Within forty-eight hours the indurated area was capped by a large vesicle approximately 2 cm. in diameter.



Fig. 6.—Case II. Robert R. Note bullous reaction to intradermal inoculation of  $1/10$  mg. O. T., and lesions on legs.

Blood: Red blood-cells, 4,680,000; hemoglobin, 76 per cent.; white blood-cells, 13,600; polymorphonuclears, 76; monocytes, 4; lymphocytes, 18; eosinophils, 2.

x-Ray examination (Fig. 5) shows marked hilus infiltration bilaterally. The shadow on the left side appears soft and mottled, with linear extension

upward and outward. That on the right side shows radiation downward to the base.

Lesions remained quite stationary and then slowly faded so that they were gone after 10/14/27. Lesions varied in intensity from time to time and appeared confluent during the last few days.

The temperature ranged between 98.2° and 100.6° F. The afternoon rise gradually subsided so that during his last weeks in the hospital it rarely went above 100° F.

He remained in the hospital from September 29th until December 22d.



Fig. 7.—Note pulmonary infiltration. This boy did not develop erythema nodosum.

His condition while in the hospital was good. After the first week he played about the ward. His weight on entrance was 18.4 kg. and on discharge 20.3 kg.

Since discharge he has been returning to the Nutrition Clinic for observation. His general condition has been satisfactory with improved appearance and steady gain in weight.

Arthur R., white boy, age two years. Brother of Robert R.  
Entered hospital 10/24. Discharged 12/22.

History of exposure to grandmother, seven weeks before her death from tuberculosis 9/15/27.

Admitted because of brother's history. He did not develop erythema nodosum but he had a markedly positive Mantoux (0.1 mg.) which, as in his brother's case, became capped with large vesicles. No complaint except perhaps stationary or loss of weight for past two months and occasional cold.

Physical examination negative except for enlarged tonsils and a few scattered moist râles posteriorly, especially on right side.

*x*-Ray (Fig. 7) showed marked increase of hilus shadows especially right, where there is seen a definitely localized triangular shadow, with extension into parenchyma of right upper and middle lobes.

Temperature irregular. Subfebrile at times with rises to 100.5° F.

Blood 11/12: Red blood-cells, 5,130,000; hemoglobin, 80 per cent.; white blood-cells, 14,600; neutrophils, 51; myelocytes, 4; lymphocytes, 35; eosinophils, 8; basophils, 2.

Weight on entrance 11.3 kg. and on discharge two months later 12.6 kg.

Virginia R., white girl, age four years. Sister of Robert and Arthur R. Entered hospital 1/12 and discharged 1/25/28.

History of exposure to grandmother, same as Robert and Arthur.

Admitted because of brother's history; underweight and failure to gain.

For the two weeks previous to entrance she had a rhinitis and irregular temperature.

Physical examination showed skin normal; the only positive findings were rhinitis, hypertrophied tonsils, a few cervical glands, marked evidence of old rickets. There were no positive pulmonary findings and D'Espine's sign was negative. Mantoux was markedly positive. She had previously been tested in September, 1927, at the time that R. entered the hospital, at which time she was negative.

Blood: Red blood-cells, 3,800,000; hemoglobin, 75 per cent.; white blood-cells, 9500; neutrophils, 56; monocytes, 7; lymphocytes, 37.

Two subsequent counts showed very little variation. Urine negative.

*x*-Ray showed increase of right hilum shadow with localized density, radiating into the lower lobe.

In this group of 3 children of the same family, all were exposed at the same time to their grandmother who died early in September, 1927. They had been in her presence for practically two months before her death. Only one of the three, Robert, developed erythema nodosum—three weeks after her death and about twelve weeks after his first contact with her. All of the children became positive to the intradermal test with 1/10 mg. Virginia, however, was still tuberculin negative at the time that Robert entered the hospital but gave a positive reaction when admitted to the hospital three months later. The two boys had a very marked infiltration at the site of the inoculation and the infiltrated area became capped with a large

vesicle. Virginia did not develop so marked a reaction. The erythema has not recurred in Robert and all of the children have been making average progress since their discharge from the hospital.

**Case III.**—Carmella C., Mexican girl, age seven years.  
Entered hospital 1/18/28. Discharged 2/13/28.

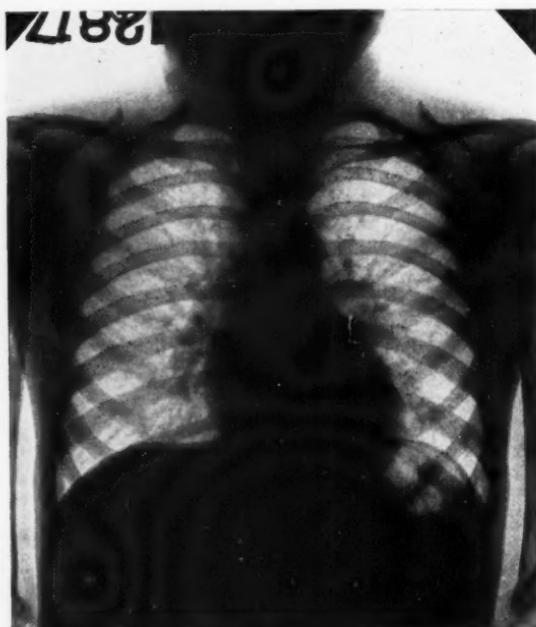


Fig. 8.—Case III. Tuberculous erythema nodosum.

*History at Entrance.*—She had an unproductive cough, which had persisted since June, 1927. Capricious appetite with loss of weight. Patchy erythema on arms and legs since 1/12/28. Fever for the previous week.

Father and mother both died of unknown cause during the year 1927. A cousin, Adolpho C., age two years, who had lived in the same house with Carmella entered the Cook County Hospital October 15, 1927, and died two days later. Autopsy revealed miliary tuberculosis. In all probability both of these children, as well as others in the family, were exposed to the same source of infection. Two other children in the family, age four and seven years, have also been infected, as well as a sister of Adolpho, who is at present a patient in the Research Hospital.

Physical examination revealed an undernourished, listless child.

Over the anterior surface of both tibiae and forearms, extending to above the elbows and knees, there were numerous dull red, inflammatory lesions  $0.5 \times 3$  cm. in diameter, with irregular, slightly raised borders. They did not fade on pressure and were very tender.

On the palmar surface of both hands there was a macular punctate, discrete eruption  $2 \times 3$  mm. in size.



Fig. 9.—Case III. Carmella C. (left): Note bullous reaction to intradermal inoculation of 1/100 mg. O. T. Anita C. (right), cousin of Carmella, lived in same home. Note intradermal reaction.

Lungs showed diminished resonance in intrascapular area, mainly on right side with sparse, fine, persistent râles in this area. D'Espine positive. Otherwise the examination was essentially negative. Mantoux 1/100 mg. showed marked induration and vesiculation. Fever rose to  $103.4^{\circ}$  F. and child appeared toxic. Repeated examinations of the blood showed a mild secondary anemia. Leukocytes normal in number, 48 per cent. lymphocytes —urine repeatedly negative.

x-Ray: Increase of hilus shadows with localized densities. No parenchymal infiltrations.

Blood calcium 11.92 mg.

About twenty-four hours after entrance, rash had disappeared. The child felt better and has continued so. Temperature has been normal since 1/29/28. Weight during stay in hospital increased from 17.9 to 19 kg.

Anita C., Mexican girl, age five. Cousin of Carmella.

Entered hospital 1/20/28. Discharged 2/10/28.

Birth and development normal. No illness until May, 1927, when a dry cough, worse at night, began, and had persisted until time of entrance. For the previous two weeks, fever worse at night, anorexia, listlessness, and night-sweats have been present.

*Physical Examination.*—Right interscapular diminished resonance and sparse scattered râles posteriorly. Enlarged cryptic tonsils, anterior and posterior cervical adenopathy. Soft systolic murmur at apex. Otherwise negative.

Pirquet markedly positive.

Blood and urine examinations were negative.

x-Ray: Average amount of lung markings.

Fever irregular—to 102° F. in afternoon.

Weight increased from 16.6 to 17.4 kg.

#### CASES APPARENTLY NON-TUBERCULOUS IN ORIGIN

While the 3 cases which we have cited undoubtedly had a tuberculous basis, it is our belief that some of the cases with which we have come in contact did not have tuberculosis as a causative factor.

We now present the history of 2 such cases and the bacterial findings of one of them, occurring on our service, which appear to be causally associated with the rheumatic group.

**Case IV.**—Victor P., Polish boy, age thirteen.

Entered hospital 2/14/28. Complained of pain over precordium, pain in abdomen, and a weakness of the legs most marked from knees to hips, less so to ankle. These symptoms had been of five days' duration. Patient had had heart trouble for four years previously. The onset followed acute rheumatic fever.

Birth and developmental history negative. Father died of heart trouble and asthma. Mother was in a tuberculosis sanitarium ten years ago. She was discharged as cured.

*Past History.*—Four years ago the child had acute rheumatic fever involving both ankles, complicated by endocarditis. The following year both ankles were again involved. Two years ago he had arthritis of the ankles, knees, wrists, and hands. Last year he had no joint symptoms and no rheumatism, but developed purplish-red, indurated swellings on both legs. The lesions were about the size of a quarter, definitely raised and very tender. This erythematous condition lasted three months and was associated with a slight fever.

Physical examination reveals the following positive findings: Nasal discharge. Chronically inflamed pharynx. Heart enlarged to the left. A loud systolic murmur at the apex practically replacing the first sound. Anterior and posterior cervical, submaxillary, left axillary, and bilateral inguinal adenopathy.

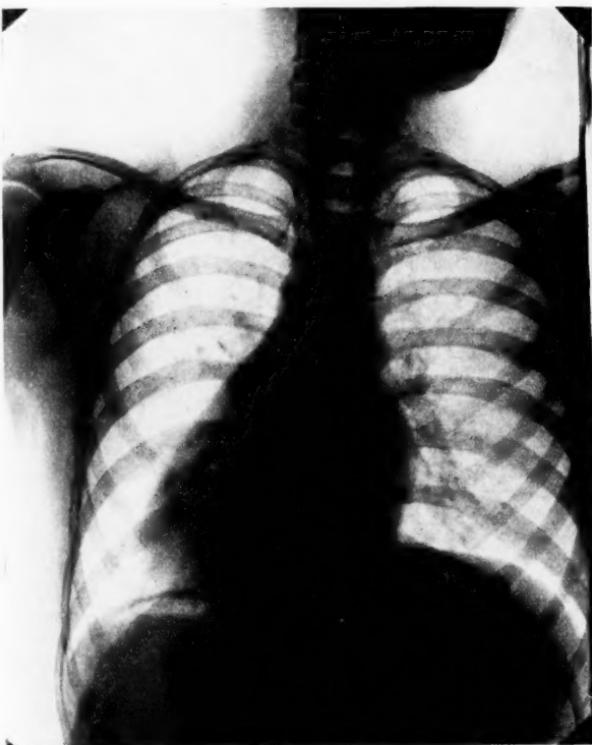


Fig. 10.—Case IV. Rheumatic erythema nodosum. Note mitral configuration of heart.

2/21: Erythematous lesions appeared on the legs, thighs, and arms varying in size from 0.5 cm. to a confluent patch  $10 \times 4$  cm. There were three lesions on the ulnar surface of the left arm, three or four on the left thigh, the large confluent patch and several smaller ones on the left leg, many small lesions on the right leg and one on the left buttock. The lesions were pink or violet in color, slightly elevated, poorly circumscribed, irregularly oval in

shape and somewhat tender. Some of the lesions showed a blanched central portion.

Wassermann reaction was negative.

Urine was normal.

Blood examined 2/15 and showed red blood-cells, 3,600,000; hemoglobin, 80 per cent.; white blood-cells, 6200.

Mantoux—done with 1/10 mg. O. T. 2/20/28—was negative 2/22/28.

x-Ray plates show a heart typical of mitral lesion. The lungs show increased hilus markings bilaterally with radiations into the peripheries. No parenchymal infiltration.

Temperature: Patient has shown an afternoon rise in temperature up to 100.6° F.

**Case V.—Mary G., white girl, age nine years.**

An attack of tonsillitis was followed in two weeks by general joint pains with definite swelling and redness only of the left elbow-joint. As the joint symptoms disappeared, there developed, without a chill, a fever of 103° F. and a crop of red, tender, painful, circumscribed subcutaneous nodes 0.5 to 2 cm., chiefly over the anterior aspect of the legs and forearms. Patient did not now (two days later) complain of sore throat. She was well nourished and well developed. The tonsils were red and enlarged, crypts filled with cheesy exudate, small superficial ulcers on the right anterior pillar; cervical lymph-glands palpable. Recovery uneventful.

At the height of the skin manifestation an erythematous node was excised from the anterior aspect of the leg. Cultures were made from this tissue, material taken from the tonsillar crypts, the ulcers in the throat and the blood, by Dr. E. C. Rosenow. From the node and blood there developed a small, usually short, sometimes clubbed, Gram-positive, non-acid fast, non-motile diplobacillus. All gradations between cocci, diplococci, and distinct bacilli could be made out in smears from each of the colonies. Those nearer the top of the tube showed relatively more coccus forms, those deeper down more bacillary forms. From the tonsils and ulcers green-producing streptococci and bacilli similar to those isolated from the blood and nodes were obtained.

Following the intravenous injection of these cultures into rabbits and a dog, beside more or less general systemic involvement, many of the animals developed subcutaneous nodules which closely resembled pathologic findings in the original excised node.

Rosenow,<sup>1</sup> in his published studies in this case and 6 others of erythema nodosum, which occurred in the wake of or were complicated by the following diseases: tonsillitis 2, rheumatism 1, pericarditis 2, endocarditis 1, and a seventh case in which tonsillitis was in all probability complicated by sinusitis, states: "As a result of this study it appears that erythema nodosum is due to a diphtheroid bacillus, closely resembling in some stages

the streptococcus group. This organism has an elective affinity for the subcutaneous tissues. The infection atrium appears commonly to be in the tonsils and pus pockets about the teeth. The pain in the cutaneous node is probably due to the fact that the hemorrhage infiltration and edema surround a relatively large blood-vessel and hence the adjoining nerve-trunk."

In looking through the literature on erythema nodosum, we are impressed with the large number of controversial publications on the subject. Erythema nodosum has been associated with rheumatism and considered due to the same unknown agent.<sup>1, 2</sup> It has been considered as a non-specific reaction to many bacterial or chemical toxins.<sup>3</sup> It has also been described as a disease *sui generis* with an incubation period of ten to fourteen days,<sup>3, 4, 7</sup> and several small epidemics have been reported in support of this opinion.<sup>5, 6</sup> Divergent views have been expressed as to the relation of tuberculosis to this independent disease. Some have denied any intimate relationship. Others, among them Wallgren, previous to 1925, stated that this disease selectively attacks tuberculous children.<sup>5, 7</sup> The skin lesions have also been considered as the response to the toxins of tuberculosis; these toxins being liberated as a result of the attack of the individual by erythema nodosum.<sup>5, 7</sup>

The causal relationship of tuberculosis and erythema nodosum was first remarked by Uffelmann and by Oehme in 1872. Since then the belief in this has gradually gained ground so that now a majority of observers support this view.<sup>5, 8, 9, 10, 11, 12, 13</sup> Some consider it as a tuberculid, and Landouzy has claimed that he was able to demonstrate tubercle bacilli in a section of the lesion. Ernberg,<sup>8</sup> in an extensive publication, has demonstrated the close association between tuberculosis and erythema nodosum. He believes the skin lesions to be autotuberculin reactions similar to an intracutaneous reaction, occurring early in tuberculosis, and due to a transient hypersensitivity of the body to the tuberculotoxin. He also found that, in about 40 per cent. of cases, manifest active tuberculosis follows in the next months after erythema nodosum.

In the last several years Wallgren<sup>5</sup> has accepted Ernberg's

views, and in recent publications quotes tuberculin sensitiveness in 135 cases in which all but 3 gave positive reactions to intracutaneous tests, in which 3 mg. was the maximum dose used. He states that the 3 cases are evidence of the fact that erythema nodosum need not be exclusively tuberculous in origin.

He further believes that there is an individual, familial, and perhaps a racial susceptibility to this particular type of skin lesion. In his experience patients who exhibit it are apt to have a benign form of tuberculosis, usually confined to the mediastinal glands.

In our own experience we have never noted the familial tendency, but wish to emphasize the fact that there may be an individual predisposition. We wish to reiterate that in the cases just presented, in one family 3 children were in our wards and in another 2 children. In each instance they were in all probability infected at about the same time and showed similar roentgenologic findings, and all of them reacted to the Mantoux tests by the development of large bullous lesions, following inoculations with 1/10 to 1/100 mg. of tuberculin.

Wallgren<sup>5</sup> states that: "Preceding the eruption a mild degree of pyrexia is constant. This may be present for only a few days or as long as a month before the eruption appears. More commonly it was of short duration. In 15 cases tested before the onset of fever only 2 were tuberculin positive, all, however, reacting positively during the fever stage."

It thus seems that the pyrexia indicates the passing of the individual from the preallergic to the allergic stage, or that the degree of allergy is speedily increasing. This onset or increase of allergy manifests itself in fever which may or may not be associated with erythema, and indicates that the body is now sensitive to tuberculin.

Wallgren further states: "Erythema occurs strikingly often in children with recent tuberculous infection. The child is infected, but is not as a rule, at the time of infection, nor during the succeeding month or months, yet allergic. Then comes the phase of the immunizing process, when the organism becomes allergic, and the moment is then ripe for the erythema. This

agrees with the fact that erythema is so common amongst children (at least in our country) and comparatively rare amongst adults. Tuberculous infection is generally contracted during childhood, and so it is during that time tuberculin allergy is expected to appear. Elderly people are as we know already allergic, or in other words they are past the period of erythema."

Wallgren also believes that a tuberculin reaction hastens or rouses erythema in those predisposed, that is to say, in children who are on the point of becoming allergic. This would correspond to the fact that a tuberculin injection, as a rule, rapidly increases the tuberculin sensitiveness in infected cases, and this consequently produces the condition necessary for an eruption of erythema nodosum, a rise of allergy.

He states: "Erythema in reality signifies that the incubation stage of tuberculosis is past, and that the eventual results of the infection may now manifest themselves."

Also, if, for any reason, an already established allergy is transiently diminished or lost, and then regained, the circumstances are again favorable to the development of erythema, namely, a change from a state of anergia or hypergia to one of allergy. Here perhaps may be one explanation why erythema is sometimes observed after an attack of measles, angina, diphtheria, scarlet fever, etc., diseases which transiently decrease more or less the degree of allergy, and also of the, with regard to tuberculous etiology, enigmatic endemic appearance of the disease in a family or school.

"If a number of children in such surroundings simultaneously incur an infection, with the capacity of decreasing their allergy when the tuberculin sensibility returns after the attack, it will be found that conditions are favorable for the increasing occurrence of erythema within a relatively limited space of time" (Wallgren).

Familial epidemics may also very likely be due to the fact that all of the children were infected at the same time from the same source, and developed their allergy and its accompanying erythema nodosum approximately simultaneously. Our R. family is an instance of a probable infection of all the children from the same source at the same time. Also Hamburger's<sup>14</sup>

2 children developed phlyctenular conjunctivitis at two days' interval, and the time and source of both their infections were the same.

"The tuberculous erythema nodosum should then be a phenomenon which sometimes appears at the moment when the organism becomes allergic, or when by one cause or another an increase of allergy takes place. The time elapsing between tuberculous infection and allergy may be considered as the real period of incubation, resembling that in acute eruptive diseases. Erythema nodosum seems to correspond to the eruption of these illnesses and ought to be considered the exanthema of tuberculosis. Contrary to the conditions in for instance morbilli the exanthema of tuberculosis is a facultative phenomenon of high degree, and cases without exanthema are incomparably more common than cases with exanthema."

Although the majority of cases of erythema nodosum, in some countries, are undoubtedly on a tuberculous basis, there still remains a small number even in these localities, in which the tuberculin reaction, with varying doses, was persistently negative. Thus, Wallgren<sup>5</sup> had 3 such cases in 135, Comby<sup>3</sup> 7 in 42, Moro<sup>20</sup> 4 in 30, Feer<sup>7</sup> cites about 400 cases described by various authors, of which 5 per cent. were tuberculin negative. Kundratitz<sup>9</sup> attempts to explain these cases in various ways. In some he insists that the test was inadequately, or not repeatedly carried out, or that a poor tuberculin was used. He also quotes Hamburger and Preyer's<sup>15</sup> observations that a tuberculin injection leads to two reaction phases, a negative with diminished, followed by a positive with increased sensibility. Ernberg<sup>8</sup> considers erythema nodosum as an autotuberculin reaction which may also lead to a transient hyposensitivity of the body. Kundratitz<sup>9</sup> also mentions the possibility that some individuals infected with tuberculosis react negatively to tuberculin due to the fact that the infection has long been inactive, and the body can overcome the irritation from tuberculin without a reaction. Magni<sup>19</sup> states that in 113 cases of rheumatism 79.12 were tuberculin negative, a much larger percentage than is found ordinarily in a similar age group. When some of these cases

developed erythema nodosum they reacted strongly positive. He mentions the possibility that rheumatism may be associated with a diminished skin reactivity, or that some children are predisposed to both diminished tuberculin sensibility and to rheumatism. He, incidentally, considers erythema nodosum a disease *sui generis*.

All of these explanations appear to us inadequate in some cases, especially in those in which the tuberculin test was repeatedly performed and the reaction was persistently negative. This is especially significant in view of Wallgren's explanation that erythema nodosum is an expression of the onset or the increase of allergy, a state where we can expect a hypersensibility to tuberculin. We are forced, therefore, to concede that a certain number of cases of erythema nodosum are on a basis other than tuberculosis. In our Case IV, there is a definite history of rheumatism, and an existing mitral endocarditis. In this case, the tuberculin reaction is negative, and it would appear that in 1927 and 1928 the erythema nodosum has been the equivalent of his yearly attacks of arthritis. In Case V also, the rheumatic origin is evident and, as has been stated, a definite organism with a special affinity for the subcutaneous tissue was isolated by Rosenow.<sup>1</sup> Robinson<sup>6</sup> also has recently reported a small house epidemic of erythema nodosum in which 3 cases were associated with fever and sore throat, and 1 had a mitral insufficiency as a sequel.

Sections of erythematous nodes show a pathology which is not specific of tuberculosis. Other toxic agents localizing in the subcutaneous tissue may cause similar lesions.

#### PATHOLOGY

The pathology of erythema nodosum reveals foci of cellular infiltrates in the deeper layers of the corium, especially in the subcutaneous fat. Insignificant and inconstant infiltrates are seen in other layers of the corium. The infiltrates are perivascular and around the glands. They consist of round cells, some multinuclear granulation tissue cells and polymorphonuclear leukocytes. In the subcutaneous fat, fibrinous exudates may

occur. The endothelium in the small vessels is frequently swollen and shows large nuclei. Occasionally small hemorrhages are found in the subcutaneous fat.

Prognosis depends on the underlying tuberculosis and other etiologic factors and not on the associated erythema nodosum.<sup>7,15</sup> This is denied by others who maintain that erythema nodosum has a deleterious effect on the tuberculosis, although some of them believe that there is no causal association between the two conditions.<sup>3, 4, 8, 14</sup> It is as yet too early to give the ultimate prognosis on all of our tuberculous cases but the first. He seems to show a very favorable course in spite of intercurrent measles, scarlet fever, and diphtheria.

The treatment is that of the underlying cause.

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## CLINIC OF DR. DON C. SUTTON

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### CORONARY OCCLUSION

CORONARY occlusion is a disease of striking interest to the student, not only because of its spectacular onset and importance as a disease, but also because of the several famous men of medicine who have died of either this or some other accident of the coronary arteries. Among these are Morgagni who reported 11 cases of rupture of the heart following most certainly infarction; Panum, who was the first to produce coronary occlusion experimentally; John Hunter, Charcot, and others. In the review of the causes of death of physicians in 1927, 125 are specified to have died of angina pectoris, not including a number of deaths from coronary thrombosis which are included under deaths from embolism and thrombosis from which cause there were 57 deaths.

It would seem that coronary occlusion and coronary thrombosis should include those cases in which as a result of slow narrowing of the arterial lumen or rapid formation of a thrombus the area of heart-muscle supplied by the occluded vessel is either almost completely deprived of its blood-supply or actually infarcted.

Cruveilhier (1850) was the first to describe coronary thrombosis. To Weigert, however, belongs the honor of describing cardiac infarction. He says: "In a very sudden cutting off of the blood-supply to the heart, yellow, dry masses similar to coagulated fibrin result."

When one considers the spectacular onset in most cases of coronary occlusion and the definite pathologic findings, it is surprising that no satisfactory description of symptoms appeared before that published by Obratzow and Strachesko<sup>1</sup> in 1910.

In 1912, Herrick<sup>2</sup> published a most important article especially emphasizing that occlusion of even a large branch need not necessarily lead to death, but that often death is postponed for weeks or months or even complete recovery may take place. Herrick's work has been followed by an increasing American literature, an excellent general review of which is one recently published by Robert L. Benson,<sup>3</sup> of Portland, Ore. The frequency of its occurrence is extremely variable in statistics. Benson and Hunter<sup>4</sup> found 14 spontaneous ruptures of the left ventricle, and a total of 72 infarcts and cardiac aneurysms in 1750 necropsies. LeCount in 175 necropsies in cases of cardiac deaths found acute coronary obstruction 26 times.

**Anatomy.**—In a monograph, *The Blood Supply to the Heart*, Gross, in 1921, by the injection of the coronary vessels, corrosion, clearing, and x-ray has carefully analyzed the circulation of 400 hearts. The most common arrangement is that of the left coronary arising from the left anterior sinus of Valsalva, slightly below the level of the free edge of the corresponding aortic cusp. Shortly after leaving the aorta it divides into the ramus descendens anterior sinister and the ramus circumflexus sinister. The ramus descendens anterior sinister passes downward in the interventricular groove to the apex, around the apex and upward over the lower third of the ventricles. Lateral branches are given off to both the right and left ventricles. The ramus circumflexus sinister passes around in the auriculoven-tricular sulcus, giving off branches to the remainder of the left ventricle especially the posterior surface. The right coronary artery supplies the right ventricle except the anterior third which is supplied by the ramus descendens sinister branch of the left coronary artery. The right coronary artery also supplies some of the posterior surface of the left ventricle and a portion of the posterior part of the interventricular septum. The anterior portion of the septum is supplied by the left coronary artery. The remainder of the septum and the papillary muscles are supplied by vessels derived from both arteries. The coronary vessels as shown by Gross are subject to great variation in their distribution. There may be only a single coronary arising from the

aorta and again there may be more than two. Of much greater importance is the question of anastomosis between the two coronary arteries and their branches. While the coronary arteries are no longer considered end-arteries, there is a great variation in the degree of anastomosis. As shown by Gross the degree of anastomosis increases with age, so that the heart of one of sixty years may be much more able to withstand the sudden occlusion of a large artery than that of a youth of twenty. This is partially to be explained by the fact that slow closure as by arteriosclerosis results in greater demands on collateral channels and results even in a rich anastomosis in extreme narrowing. At times, also, the heart-muscle may receive some nutrition through the thebesian vessels, the arteria<sup>e</sup> telæ adiposæ (distributed in the epicardial fat) and rarely through anastomosis with vessels of the mediastinum at the base of the heart.

The ramus descendens sinister is of especial interest because it is the vessel most frequently involved in coronary occlusion, being the artery involved in perhaps four-fifths of the cases. The left coronary is involved seven or eight times more frequently than the right.

In 1884 von Leyden (see Benson<sup>3</sup>) in classifying cardiac injury from coronary obstruction, distinguished four groups:

1. No anatomic change, death occurring so suddenly that time is not allowed for anatomic changes.
2. Myomalacia cordis (infarction).
3. Slower progressive closure of artery producing myofibrosis, either in disseminated islands, or in large confluent areas of fibrosis, or in the form of aneurysm.
4. Mixtures of these.

When an artery is occluded by a thrombus or embolus there may or may not be an infarct formed, depending upon the degree of anastomosis present. If infarction occurs its area is always smaller than the distribution of the artery involved. Experimentally when an artery is first occluded the area of distribution becomes pale, then cyanotic. Usually at postmortem the infarct appears as a yellowish area. Its resemblance to fat led the older observers to describe infarcts as areas of fatty de-

generation. The area of infarction is often surrounded by a zone of hemorrhage. If the endocardium is involved thrombi may form from which may originate emboli to various parts of the body.

Hemorrhages are often seen in the pericardium over the infarct, and localized fibrinous pericarditis is frequent, giving rise to one of the characteristic physical findings. The area of infarction is not always visible macroscopically as first noted by Marie, but is found on microscopic examination if the suspected area is as in the case cited later. The infarct may heal, rupture may occur, or aneurysm formation with later rupture may result.

**Etiology.**—*Age.*—Coronary occlusion very seldom occurs before forty years and, as a rule, occurs after fifty-five years with most of the cases falling between sixty and seventy years.

*Sex.*—It is about twice as frequent in males as in females.

*Arterial Disease.*—Arteriosclerosis and, as a rule, hypertension are almost constant, indeed, it would appear necessary that at least localized coronary sclerosis be present in virtually all cases.

*Syphilis* is very rarely a cause, and when it is, is due to involvement of the aorta at the openings of the coronaries.

*Diabetes*, in which of course arteriosclerosis is very frequent, has been present in a number of my own series.

**Symptoms.**—The characteristic feature of the symptoms of coronary occlusion is their sudden onset, but it is well to remember that the occlusion may be so gradual that there are no symptoms other than increasing myocardial weakness, or the condition may occur in the course of other cardiac disease when there may only be an increase in signs of failure, and infarction is only suspected by the suddenness of the change.

Herrick divides coronary occlusion clinically into four groups:

1. Cases in which death is sudden.
2. Cases in which severe anginal pain and profound shock is followed in a short time by death.
3. Mild non-fatal cases which are difficult or impossible to recognize.

4. Cases in which symptoms are severe, which may or may not be fatal.

The onset is often precipitated by strain, overeating, or emotion, but may occur in the midst of apparent health and during a period of rest. When a history of myocardial disease shows a sudden onset coronary occlusion should be considered, especially if ushered in with pain. Pain is the most usual ushering symptom, anginal in character, but more prolonged (the status anginosus of older authors). The location and distribution are the same as in angina, namely precordial and substernal, often more boring in character, referred to the neck, down the left arm, and more often down the right arm than in angina. I have seen the pain felt only in the interscapular region. The pain may last for hours or even days, and be unrelieved by morphin. Pain in the abdomen is frequent and at times it is the only location of pain. It may be accompanied by nausea, vomiting, tenderness, and rigidity in a striking imitation of an accident in the upper abdomen, which not infrequently leads to the fatal error of surgical intervention.

*Dyspnea* is usually a prominent symptom, and especially so when pain is absent, as in the case quoted.

*Shock* is always marked. The face assumes an anxious expression, has a peculiar pale gray appearance without cyanosis. Beads of perspiration stand out on the forehead and the pulse is weak, even at times imperceptible. There is usually marked fall in blood-pressure, often below 100, and is considered one of the diagnostic signs.

**Physical Findings.—Heart.**—Insofar as I know, this is the only condition which produces an acute dilatation of the heart. Coming on as the attack so often does after overstrain or a heavy meal, this dilatation is likely to be interpreted in the light of the apparent cause and not as due to a thrombosis. The pulse is usually rapid and weak, although in occasional cases, it may be normal or slowed. The heart borders are usually increased especially to the right. Instead of the heaving tumultuous heart action expected, as implied by the symptoms, there is no precordial activity, and often even the apex is imperceptible.

The tones are usually distant and weak. Existing murmurs may be unaffected and a systolic murmur may be first heard after the accident. Irregularities of conduction are frequently present. In experimental ligation of the coronary arteries ventricular fibrillation is often induced. This may be the reason for some cases of sudden death. Premature contractions are frequent, as may be the occurrence of paroxysmal tachycardia of ventricular origin. Auricular flutter is occasionally seen and auricular fibrillation is rather frequent.

*Pericardial Friction.*—Herrick stresses the importance of pericardial friction as a diagnostic point. On the other hand, Wearn noted it only twice in 19 cases, and Loncope not a single time in 16 cases. As for myself, I have not failed to find it in any case I have been able to see at frequent intervals from the beginning. Like other pericardial friction wounds, it may be transient and heard only once or twice during the course. When the infarct is on the anterior wall, as is the rule, I believe if carefully and frequently looked for, it will always be found. The earliest I have heard it was twelve hours after the onset, but the usual time is during the interval from the second to eighth day. Its presence is always confirmatory of the diagnosis.

*Pulmonary edema* is such a constant finding that it may be considered one of the important diagnostic symptoms. There is always edema of the bases as evidenced by moist râles and rarely there may be an acute pulmonary edema. Small amount of serous or serosanguinous fluid may be expectorated.

Fever is rarely absent and usually begins after twenty-four hours. It often is 100° F., but rarely may reach 102° F. Usually it subsides in from four to ten days, but occasionally may last for weeks. Leucocytosis of from 10,000 to 15,000 is the rule. The finding of fever and leucocytosis is often held in favor of abdominal lesion when the pain is abdominal unless, one is familiar with their presence in coronary occlusion.

*Embolic Phenomena.*—If the infarct involves the endocardium it is the rule that thrombi form within the ventricular cavity. Emboli are frequent to the lung with pain and hemorrhagic sputum if the thrombus is in the right ventricle. To the

kidneys, spleen, brain, and other peripheral arteries if the left ventricular wall is involved. Emboli usually occur after an interval of several days and are especially valuable as a retrospective aid.

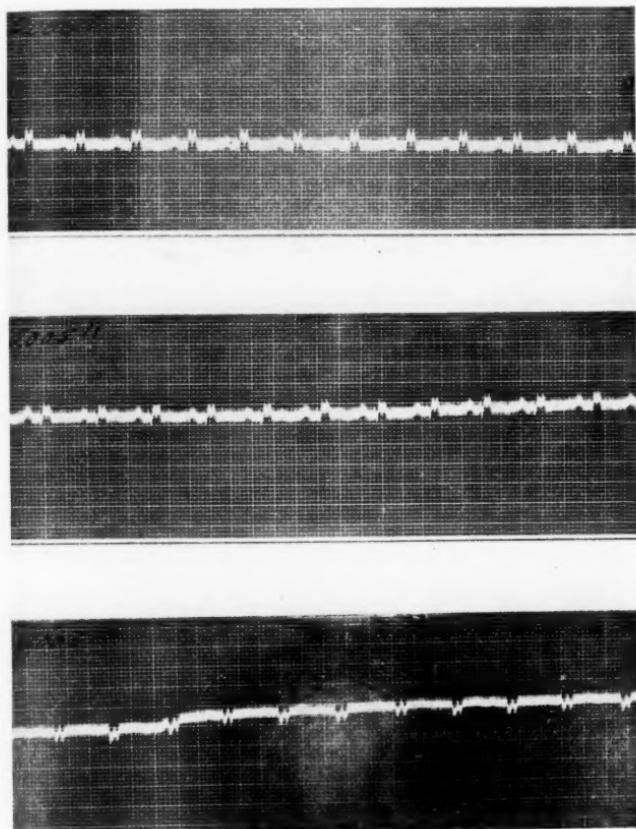


Fig. 11.—Electrocardiogram showing disturbed conduction in arborization tissue. From a case of coronary occlusion proved at necropsy.

Cheyne-Stokes respiration is often present in severe cases.

One of the late results of coronary occlusion is cardiac aneurysm usually located at the apex, not often recognized, but may be

rarely suspected by a bulbar outline at the apex or by its outline in an *x*-ray. It is usually discovered at necropsy following rupture.

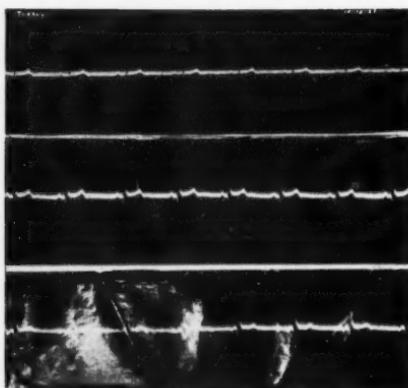


Fig. 12.—Electrocardiogram taken the day following attack. Note low-voltage Lead I. The T comes from high up on the down stroke of R in Lead II from Case II.

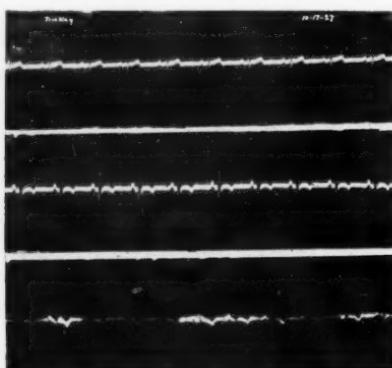


Fig. 13.—Electrocardiogram from Case II taken the fifth day after onset. Note inverted T Leads II and III.

*Myocardial Disease.*—Usually the course of coronary occlusion is that of the condition commonly known as chronic myocarditis.

Many cases may go for years with well compensated hearts, but the usual result after a variable period is myocardial failure.

*Electrocardiogram* may be of utmost importance in the diagnosis of this lesion when typical. On the other hand, many of these cases show only the changes that are usually attributed to myocardial damage or to disturbance of conduction in the Purkinje system. These latter changes are lowering of voltage in the Q.R.S. with deformities in this complex as notching and slurring of the R (Fig. 11). Fred Smith has recorded by experimental ligation the typical electrocardiogram. Early and constantly the beginning of the T arises from the down stroke of the R (Fig. 12). The T is usually inverted in one or more leads, especially Lead II (Fig. 13).

The following histories will serve as typical examples of coronary occlusion:

**Case I.**—Dr. E. M. C., female, age fifty-eight years, was seen in consultation forty-eight hours after the onset of the illness. After eating a very hearty dinner and while at rest, during laughter, was suddenly seized by an extreme dyspnea. She was seen within fifteen minutes by her attending physician, Dr. Mary McEwen, who thought her in extremis. Sedatives were given immediately and she was removed to the hospital at once. On the way to the hospital the ambulance was stopped as it was thought the patient was dying, and additional medication was given. During this period the pulse ranged from 120 to 160, was absolutely irregular and at times imperceptible. Under sedatives and digitalis she became more quiet and by the next day the pulse was slower and better. Although the dyspnea continued it was much less severe. Six hours after the onset she complained of severe pain over the precordium and referred to the right shoulder and the right arm. This pain was continuous for three days. On the second day she complained of pain in the upper abdomen, which because of the coincident temperature and leukocytosis caused considerable concern as to a possible acute lesion of the abdomen. When seen at this time she was having moderate dyspnea and orthopnea and was moderately cyanosed.

The left heart border in the fifth intercostal space measured 15 cm., and the right border in the fourth intercostal space, 4.5 cm. An old mitral disease was present, dating from a chorea at fourteen years of age. An auricular fibrillation was present. In the fifth intercostal space at the sternal border a pericardial friction was present. Her blood-pressure on entrance was 140 mm. systolic and 80 mm. diastolic; during examination it was systolic 120 mm. and 70 mm. diastolic.

White blood-cells 14,800.

Temperature 101° F.

On the fourth day she felt perfectly well and was planning her return home. She suddenly awakened from sleep with an agonizing precordial pain, and within three minutes was in coma. Six hours later she died.

At necropsy an embolus was found occluding the internal and left common carotid arteries. A small area of fibrinous pericarditis was found in the pericardium near the apex and also an area of subpericardial hemorrhage. No other evidence of coronary occlusion was found grossly. After fixation of the heart a thrombus was found in the ramus descendens sinister. Microscopically a hemorrhagic infarct was found (Fig. 14).

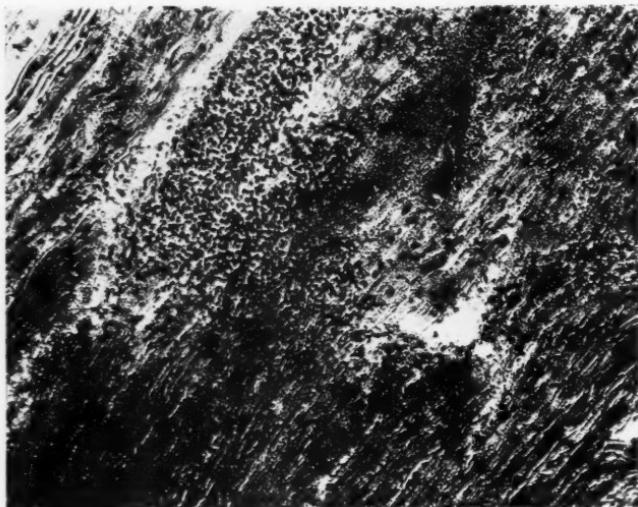


Fig. 14.—Microphotograph of hemorrhagic infarct found in Case I.\*

**Case II.**—Mr. J. T., age forty years, was first seen on October 10, 1927. He had been perfectly well for months preceding the attack. When on his way home from the office he felt a sense of oppression over the midsternum. Within the two blocks he had to walk to his "L" station the substernal pain became so severe he began to fear he could not get home. However, he managed to walk up the elevated stairs and get on his train. During the trip of forty-five minutes on the train the pain was almost unbearable but he managed to walk to his home two blocks from the station and up a flight of stairs. As his home is near mine I was able to see him within ten minutes after his arrival home. He was lying on the bed writhing in pain, his face was drawn, ashen gray, and covered with beads of perspiration. (Note: A patient with an angina would have stopped with the beginning of the pain, the duration

\* Illustrations are by Miss Walsh of the Photographic Department of Northwestern University Medical School.

would have been shorter, and if in bed during an anginal attack the patient lies perfectly still, but in coronary occlusion the patient is constantly trying to find a position of comfort.) The pulse was 60 but small, the heart moderately enlarged. No murmurs nor friction, tones were distant. He refused hospitalization so was given  $\frac{1}{2}$  gr. of morphin hypodermically. An hour later he asked to be taken to the hospital and there received morphin (gr.  $\frac{1}{4}$ ) every two hours all night and part of next day when the pain disappeared. In eighteen hours his white count was 12,800. A pericardial friction was evident at the end of twenty hours and persisted for three days. On the third day his temperature rose from normal to 100.2° F. The next day it was 100.2° F. and by the seventh day was normal. His pulse continued normal except on the eighth day when he had an attack lasting thirty minutes, during which the pulse was counted at a rate of 160 by the intern. As this attack began and ended suddenly we may presume it was an attack of paroxysmal tachycardia. Otherwise the course was uneventful and after one month in bed and two months in his apartment he was allowed to get out slowly. He is now back working from twelve to fourteen hours a day as a salesmanager.

**Treatment.**—The treatment of the condition can almost be stated in one word—*rest*. Rest as nearly complete as possible is of the greatest urgency and should be continued for as long a period as possible. Certainly not less than one month bed rest is necessary for the healing of the infarct and several months are required for complete recovery. Morphin is essential during the period of pain and quite large doses may be required to give even partial relief. Digitalis is contraindicated because it increases irritability and should not be used except in instances of auricular fibrillation. In these instances its specific action is more important than its effect on irritability.

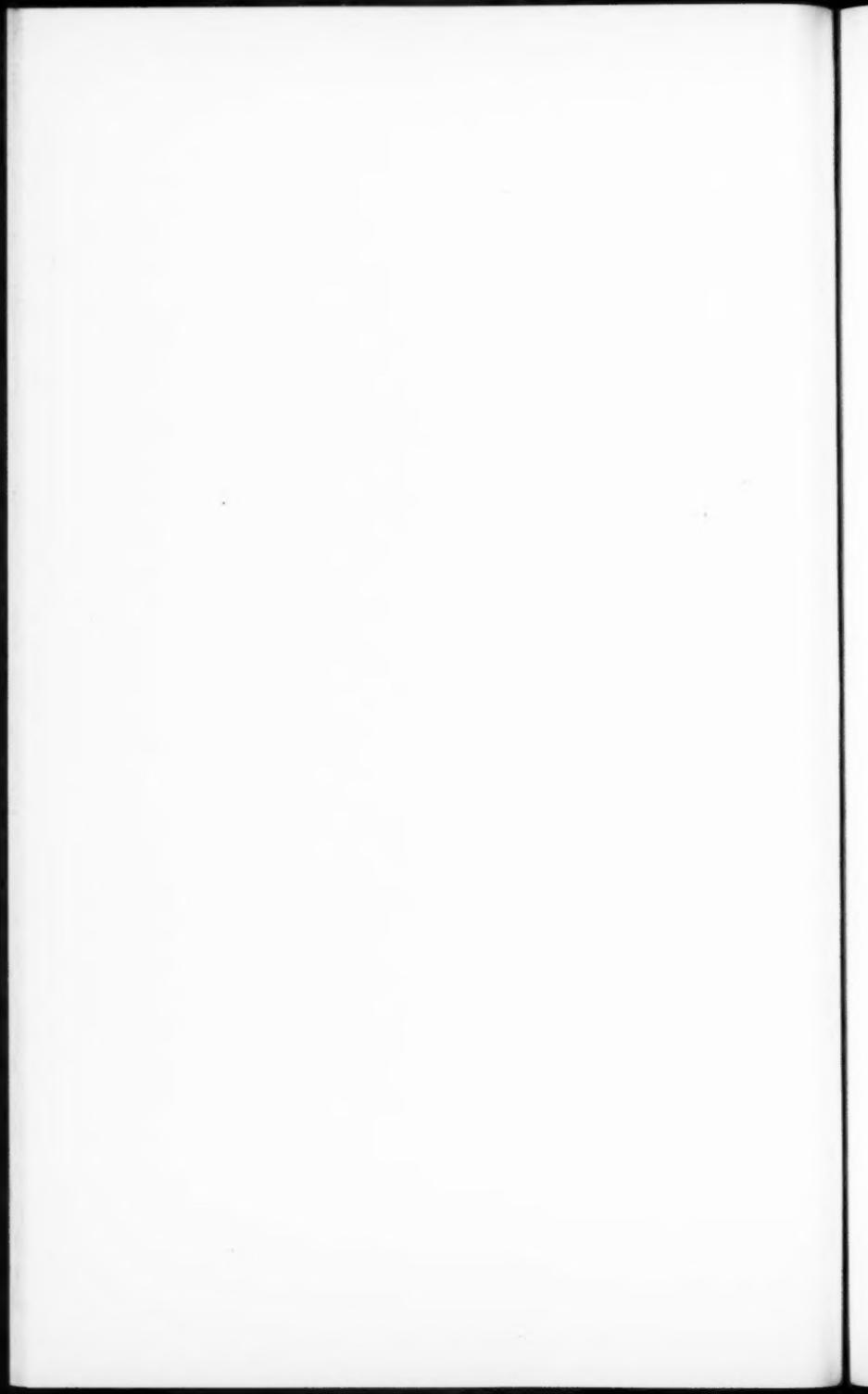
**Summary.**—Pain, anginal in character, lasting for several hours is alone sufficient to diagnose coronary occlusion.

Pain, dyspnea, shock, pericardial rub, and changes in electrocardiogram are the cardinal manifestations.

Treatment is essentially bed rest for a long period of time.

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CLINIC OF DRs. SOLOMON STROUSE AND  
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**DIABETES. LATE RESULTS OF INSULIN TREATMENT.  
TREATMENT WITH SYNTHALIN**

You will recall that insulin was introduced in the spring of 1922. Since then it has been accepted as a standard remedy in the treatment of diabetes.

From clinical observation we have long been of the opinion that the administration of insulin over a long period of time results in a raising of the individual carbohydrate tolerance. A short survey of the literature shows, however, a distinct division of opinion. McLeod and Campbell<sup>1</sup> have observed experimentally in partially depancreatized dogs that the administration of insulin causes maintenance of excellent nutrition for from two to seven months; then occurred an acute breakdown in liver function and death despite the use of the pancreatic hormone. However, they have also noted that in some animals the dosage may be reduced from time to time—due to the release of a latent tolerance submerged by previous overwork of the pancreas. Whatever the cause, we have here indisputable evidence of an increase in tolerance after the administration of insulin. From pathologic study of 1 case, Boyd and Robinson<sup>2</sup> conclude that there is a regeneration of islet tissue. Clinically their nine-year-old patient showed an increase in carbohydrate tolerance of 45 gm. in seven years. Allen and Opie, both, in discussing the case, state that there is evidence of hypertrophy of island tissue. McCallum has made a similar observation.

Clinically the reports are at variance. Newburgh,<sup>3</sup> from a study of 3 cases which seemed progressively to lose tolerance, concludes that "at present there is no published evidence that

insulin increases the tolerance of diabetes." LeFevre<sup>4</sup> after study of 57 cases, half of which required insulin, noted that half of those receiving insulin were able to discontinue it in less than fourteen months provided that the drug was given in such dosage as to keep the fasting blood-sugar approximately normal. Harrison<sup>5</sup> observed 5 cases for from eleven to eighteen months; he reported a virtually stationary tolerance, but thought that variation in the strength of the insulin used was such that a definite conclusion could not be formulated. Brace<sup>6</sup> watched 5 patients for from thirty-two to forty-five months and "has no ground for believing that insulin is capable of effecting a cure or a partial cure of human diabetes mellitus." By far the most convincing study of an opposite kind is afforded by Leyton. He divided his patients into two groups: (1) Those able to afford, from the inception of treatment, all the insulin required to keep the blood-sugar constantly below 150 mg. per cent.; (2) those able to afford only enough insulin to prevent glycosuria. The first group required less insulin as time passed; the second, on the other hand, required more. It is doubtless too early to tell definitely whether insulin increases the carbohydrate tolerance of the diabetic patient, but the study seems well worth while for very obvious reasons. We are interested in seeing what a few of our patients, who had received insulin for a long time, are doing, and for that purpose we will present today some records.

**Case I.**—This patient is a woman who was first seen on February 13, 1922. At that time she was thirty-seven years of age. She gave a rather acute history dating back to September, 1921. However, more careful analysis showed a loss of weight during the preceding three years from 150 to 132 pounds. The more recent story was her inability to remain sugar-free except on a very low diet. We estimated that her diet then contained protein 110, fat 90, and carbohydrate 64. In a month she lost an additional 4½ pounds. Sugar and acetone were present almost continuously. In December, 1922 it is noted that the weight loss had continued and the urine contained 36 gm. of sugar, much acetone and diacetic acid.

About this time we were able to get Lilly's experimental iletin, the exact dosage of which at that time was not completely worked out. The glucose value of her diet was then about 54 gm. We gave her in the beginning what we thought was 10 units daily. To those who were not working in those dramatic days it might be of interest to mention that the original clinical experiments were conducted not always under perfect conditions.

Even with the full co-operation of the Eli Lilly Company it was at times exceedingly difficult to know how much insulin was being used.

By April, 1923 she was taking 15 units twice a day and was on a diet containing approximately 95 gm. of glucose. The urine was negative, her weight 134½ pounds, and she clearly showed a very remarkable improvement. Then she broke all rules, and by September, 1923 confessed that she was eating almost everything and taking 30 units of insulin twice daily. She weighed 137 pounds. By that time she knew how to balance her diet and insulin and could keep herself sugar-free whenever she wished to. From then until the present time her condition has been one of steady clinical improvement. She continued to gain weight until in February, 1926 she was up to 150 pounds. She would not keep on an accurate diet, but, as said before, varied her diet. Her blood-sugar during this period varied from 200 to 235 mg. per 100 c.c. of blood. The urine would vary depending entirely on the diet.

By December, 1926 she began to show very marked edema of the hands and feet which with the weight changes apparently bore a direct relation to the carbohydrate metabolism and to the amount of insulin taken. When she became sugar-free her weight would immediately drop. When she allowed herself to be careless and became loaded with sugar, her weight went up and edema developed. On October 5, 1927 hospital check-up showed a blood-sugar of 222. It also showed that she could remain sugar-free on a diet of protein 75, fat 100, and carbohydrate 50, with 20 units of insulin twice a day. Since then her condition has remained unchanged.

*Discussion.*—This patient represents anything but a controlled experimental study. She does represent a condition met with in private practice frequently enough, namely, the diabetic who refuses to lead a strict life and who will take insulin for the sake of the comfort and convenience and a better nourished condition. Spirited academic discussion has very little value in contrast with the apparent fact that this lady's life was changed from a steady down-hill progress to six and one-half years of enjoyment of real living. Her metabolism certainly leaves much to be desired, yet she has been able to live her own life in her own way. After all, a physician is more or less of a philosophic guide to his patients, and if by the use of insulin he can assist a patient in working out his way of salvation, we do not see why we have to decry the inability to do a perfect job.

This case leaves unanswered the question as to the effect of insulin on carbohydrate tolerance, because the insulin was not

given a square deal. But it answers very satisfactorily the broader question of life and its enjoyment.

**Case II.**—This patient was fifty-three years of age when first seen on October 9, 1923. The duration of the diabetes was one and one-half years, during which time she had lived on a strict diet and had no subjective symptoms and no loss of weight. She came to see us because "she had sugar in the urine."

She was a very small, thin woman with a negative physical examination. The urine contained a trace of sugar. Her blood-sugar was 214 mg. She evidently had not been feeling well before coming to see us.

Without any great change in diet, she showed increasing acidosis for which hospitalization was advised. She entered the hospital on October 16, 1923, weighing 109½ pounds. In the hospital her blood-sugar was 238 to 246 mg. and non-protein nitrogen was 44. When she left the hospital her diet consisted of protein 75, fat 150, carbohydrate 65, and she was taking no insulin. She went home feeling well. Then she began to demonstrate a characteristic which was the exact opposite of the one we just commented on in the first patient; namely, she did not eat enough food and, as a result, soon showed clinical signs of acidosis with much acetone in the urine. After considerable persuasion she consented to eat more but did not really live up to her promise. By July, 1924 she had stopped taking insulin, for some time had weighed only 102 pounds, and showed sugar more or less all the time.

She disappeared for a year and returned in July, 1925, looking like a skeleton, weighing 94½ pounds, with the frank admission that she was eating very little and was under a severe nervous strain. Despite this, the urine showed some sugar and again insulin was advised. Again she did not follow the advice to eat more, and she returned in September, 1925, weighing 94½ pounds, with the urine loaded with sugar, acetone and diacetic acid. We started insulin in October, 1925, although there was some doubt as to the amount given. She began to show considerable improvement, and by February, 1926 her weight had gone up to 100 and she was feeling much better, but the urine still contained sugar. Then she relapsed into her mania of not eating enough, and although she was taking 40 units of insulin twice a day during all this time, she steadily lost weight and the urine contained much sugar. We were unable at that time to make a complete metabolic study but it looked very much as if she were eating so little that she was burning her own body. On September 20, 1926 she was picked up in a downtown store in what was supposed to be a cerebral apoplectic stroke and was sent to another hospital. She recovered completely from the "stroke" which was interpreted as being due to delayed insulin reaction; for on questioning it was learned that on this morning instead of taking 40 units she had used 80 units and had eaten very little breakfast and no lunch. From that day on the patient's mental attitude toward food was changed and for the first time we were able to make her eat enough. As a result it was soon apparent that she could take much less insulin and improve clinically. In March,

1927 she weighed 115 pounds, the urine was negative and she stated that she was feeling fine. She was taking 30 units of insulin twice a day.

By May she was taking only 25 units twice a day, still eating well, and weighed 116½ pounds. From May, 1927 to February, 1928 she has varied her diet and the insulin somewhat but has felt very well, and during the past winter has overcome an exceedingly severe respiratory infection involving the nose, throat, and bronchial tubes. At the end of this infection she was only taking 15 and 10 units or even 5 and 10 units a day. The urine remained sugar-free and she weighed 112½ pounds. As far as we can determine she is taking all the food she needs, but she does not weigh it and the exact figures are therefore lacking.

*Discussion.*—Again we have an example of an individual who does not follow rules. In contrast with the previous patient she underate rather than overate, but at the end of five years' treatment she is considerably improved. You will recall that her minimum weight was 93 pounds. Her weight today is 112½ pounds (February, 1928). She is able to lead a perfectly full normal life. Although we have not exact statistics to prove it, the data we have indicates that in the course of these five years there has been an increase in her tolerance. Whether this is due to the insulin or to her rather long period of undernutrition, we of course cannot say; but the probabilities are that the undernutrition was a more potent factor than the insulin.

We will briefly mention a few other cases of interest.

**Case III.**—This young man (now aged thirty) was first seen September 21, 1921 with a very severe juvenile diabetes. In June, 1922, on a diet of protein 52, fat 100, carbohydrate 28, he was passing sugar and had a blood-sugar of 300 mg. per 100 c.c. of blood. At the present time, this young man is actively engaged in business in California and leading a perfectly normal healthy life. We are told that his diet consists of 2400 calories, containing a glucose value of 250 gm. He is taking 80 units of insulin. The last time the patient was seen by one of us (S. S.) he was the picture of health and had none of the signs that we would expect of him had he remained untreated with insulin.

**Case IV.**—This young woman first consulted us in August, 1924, at the age of twenty-five years. She gave a history of diabetes of three years' duration, apparently rather mild until two and one-half months previous. At that time she took insulin for a week, discontinued it and remained sugar-free until about a month before we first saw her. At that time she was a rather frail young woman, showing nothing of importance on physical examination but with the urine containing much sugar. She did not do very well on office control and was admitted to the hospital.

She continued to take 20 units of insulin twice a day on leaving the hospital and by November, 1924 her diet was protein 65, fat 150, and carbohydrate 65, and she was using 10 units of insulin in the morning and 5 in the afternoon. However, there was some sugar present. Insulin was increased. Control was not very good until February, 1925, when she seemed to have struck a balance and remained sugar free on insulin dosage varying from 20 units to 40 units twice a day. On October 12, 1925 she developed a boil on the buttocks, for which she was admitted to the hospital in a state of precoma. The abscess opened spontaneously and gave no further discomfort. Usual diabetic management cleared up the acidosis and the patient was discharged from the hospital on the fourteenth day with a diet of protein 34, fat 142, carbohydrate 39, and 10 units of insulin three times daily.

Not satisfied with the troubles that she already had, she became pregnant and was readmitted to the hospital and curedtted. In April, 1926 her weight was 122 pounds and she was sugar-free, taking 20 units of insulin twice a day. From then until the present time (February, 1928) her general health has steadily improved and the urine has remained perfectly free from sugar. Her weight has gone up to 135 and she is taking 10 units two or three times a day depending on what she eats. Her diet is well balanced and she is happy.

**Case V.**—Only one more patient will be mentioned, a woman who was seen originally on September 8, 1921, at which time she was forty-seven years old. She gave a history of having had diabetes for ten years. She was an advanced diabetic showing marked changes of diabetic retinitis with old and new hemorrhages, a blood-pressure of 200/100, a urine showing a specific gravity of 1030 with a large amount of sugar but no other findings. The blood-sugar was 400 mg. to 100 c.c. of blood and the urea nitrogen was 33 mg.

It is rather remarkable to be able to report almost seven years later a condition as follows: General condition excellent. Weight is 130 as compared to about 108 when first seen. She is taking 20 units of insulin once a day and up to quite recently was sugar-free. About a month ago while in Canada she had an infected finger and the doctor, finding a rather high blood-sugar, put her on a diet of protein 50, fat 200, and carbohydrate 50, with 20 units of insulin once a day. We saw her on February 22d after an absence of over a year. She weighed 130 pounds. Her blood-pressure was 180/70. There were no fresh hemorrhages in the eyes and her general condition was excellent.

This patient illustrates one important point which should be emphasized regarding the use of insulin. In 1921 she was already beginning to show signs of cardiovascular degeneration which is so common in diabetics of the fifth and sixth decades, and which makes treatment even more difficult than that of uncomplicated diabetes. With insulin we were able to keep the

diabetes under control on a diet which certainly did not harm the kidneys with the result that there was no down-hill progress in almost seven years.

### SYNTHALIN

The use of synthalin which was advocated by Frank a short time ago is causing considerable interest. We have been doing some experimental work with this drug and have employed it on a carefully planned basis in the treatment of some cases of diabetes. Opinion seems to be divided as to its value. We will show our patients and let the results speak for themselves.

**Case VI.**—This patient, a male, aged sixty-three, has had diabetes for six years. He entered the hospital with marked undernutrition, moderate arteriosclerosis, weighing 109 pounds. On a diet of protein 60, fat 100, carbohydrate 40, and no insulin he maintained a more or less constant daily sugar excretion of 30 gm. He was then given 50 mg. of synthalin daily with only a negligible reduction in his sugar excretion. Increase of the drug to 100 mg. daily was immediately accompanied by anorexia, malaise, nausea, and vomiting. Of course he did not eat and the sugar excretion immediately diminished. The blood-sugar during this time fell from 234 mg. to 197 mg., and his weight dropped to 102 pounds. We naturally felt that the change in his blood-sugar and the reduction of his urinary sugar were unquestionably due to the very marked undernutrition which took place as a result of the intoxication by synthalin.

After a few days' rest, he was again able to eat the diet given above and the synthalin was begun on a schedule of 50-25-50-0 mg. daily. The sugar excretion varied between 6 and 60 gm. per day. Because of weight loss the diet was increased to protein 60, fat 180, carbohydrate 85. He was then given insulin in order to make him sugar-free. The blood-sugar dropped to 156 mg. He was then given synthalia in the dosage just outlined in addition to the insulin. The insulin was gradually reduced, but not stopped, and the patient remained sugar-free; then the synthalin was discontinued. He continued sugar-free until the insulin was further reduced.

This case represents two experiments, one to see if with the steady excretion of a comparatively small amount of sugar, synthalin reduced the amount of sugar excreted, and the other to see if we were able to replace a certain amount of insulin by synthalin. It would seem that in neither experiment was the drug efficacious.

**Case VII.**—A woman, aged forty-nine, had for two and one-half years a rather mild diabetes. No insulin had been given. Physical examination showed moderate overnutrition, slight cardiac hypertrophy which was apparently due to overactivity and which cleared up after rest and small doses of digitalis. On a steady diet of protein 75, fat 175, carbohydrate 140, there was a daily sugar excretion of 10 to 15 gm. This certainly places her in the group of mild diabetics. Synthalin was given on a schedule of 50-25-50-0 mg. daily, but there was no difference in the sugar excretion. On the contrary, insulin was given one day and the urine-sugar immediately cleared, but reappeared the following day and was again uninfluenced by synthalin. With a very slight reduction of dietary intake the glycosuria disappeared and the patient left the hospital in good condition.

Again it would seem that in a very mild diabetes synthalin is without effect.

**Case VIII.**—A male, aged thirty-six, had had diabetes for four or five years. Physical examination was practically negative except for pyorrhea alveolaris. He was made sugar-free by diet and insulin. He was given synthalin on a schedule of 50-25-50-0 mg. daily, and the diet was increased by 20 gm. of carbohydrate. He remained sugar-free. It seemed possible in this case to use synthalin in place of insulin. However, we discontinued the synthalin and the patient remained sugar-free but when we reduced the insulin to 5 units glycosuria promptly appeared. It disappeared when the insulin was raised.

Here we have a case with what looked like a synthalin effect, but it does not bear inspection too closely.

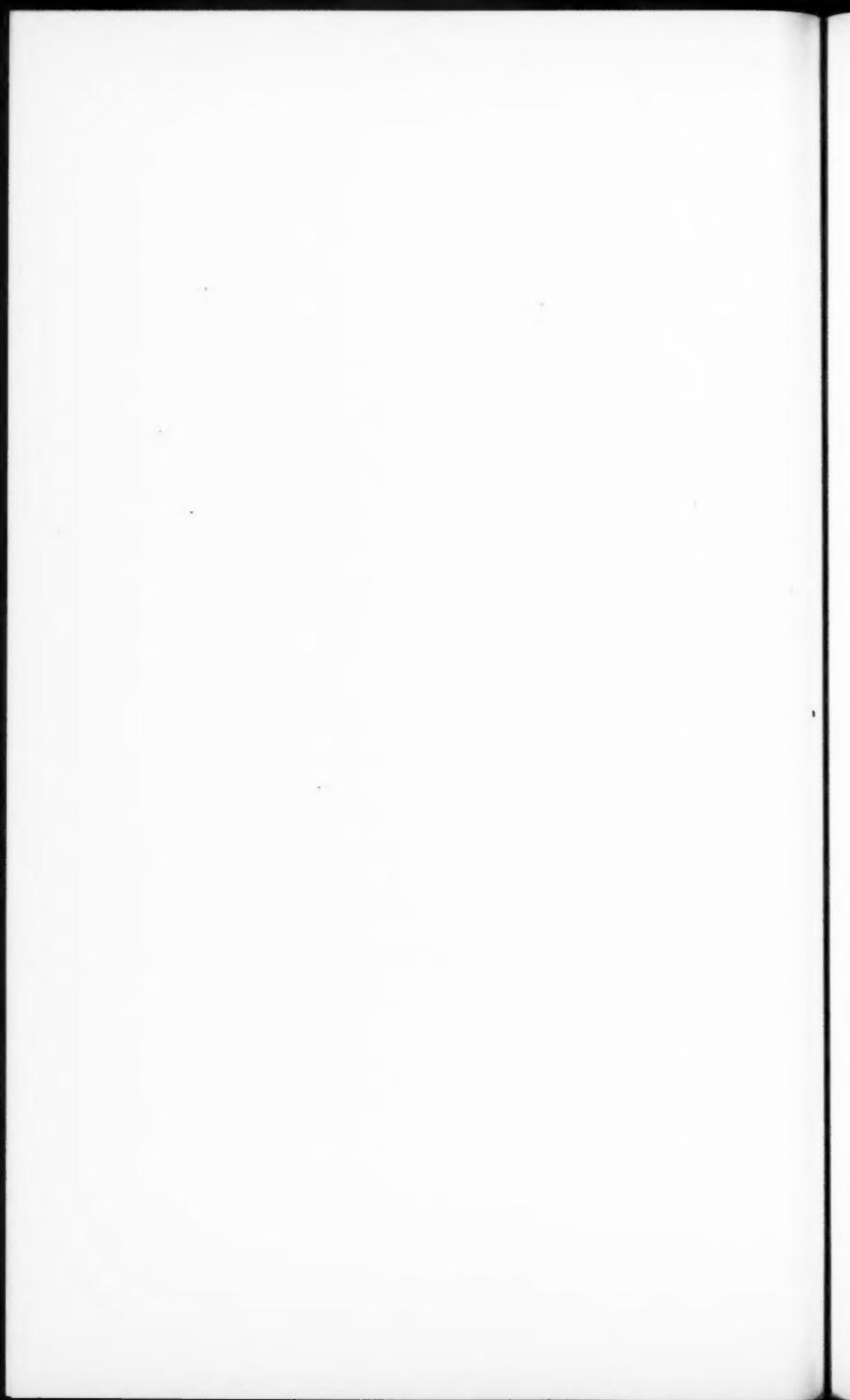
**Case IX.**—This case we will not discuss in detail. The patient is a woman, twenty-nine years of age, with a most unusual type of diabetes if it can be called diabetes. Her symptoms date back five months and were accompanied by 40 pounds gain in weight. Physical examination showed marked obesity of the so-called pituitary type. There was no x-ray evidence of pituitary tumor. The basal metabolic rate was +15 per cent. The blood-sugar was 232 mg. on a diet of protein 70, fat 160, carbohydrate 60, there was a constant glycosuria of 5 gm. daily. On synthalin in doses of 50-25-50-0 mg. daily there was no change in the sugar excretion. She became sugar free with rigid dietary reduction.

In addition to these cases there are two in whom the synthalin produced such toxic symptoms, nausea, vomiting, and abdominal pain, that it had to be stopped. One patient was given only 25 mg. a day for five days and the other patient was given 25 mg. for two days, and 50 mg. for two days.

**Discussion.**—We do not wish to draw conclusions from this small series of cases, but it is clear from this report that we are dealing with a substance which may produce very marked toxic symptoms. The work of Szezeklik and Iversen and Munck would indicate that these toxic symptoms are not to be cast aside lightly as they may indicate more or less serious derangement of the liver or kidney. In the patients who were able to take the drug it would seem from our results that it was without particular value. It is not at all difficult to make conclusions fit a preconceived idea in the treatment of diabetes as the variations in the disease may be marked. We are not trying to prove or disprove anything except whether synthalin works. In order to do this we set standards of control which we believe to be essential in the interpretation of any form of diabetic therapy. Employing such a basis for study we are forced to the conclusion that at the present time our series of cases does not justify the belief that synthalin is a valuable drug.

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## CLINIC OF DR. JAMES G. CARR

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### CLINICAL SIGNIFICANCE OF THE ELECTRO-CARDIOGRAM

WITH the introduction of the electrocardiograph into clinical medicine, it was hoped that this new means of instrumental examination would be an important aid to diagnosis. To a great extent, these early expectations were not met. The new method immediately proved valuable in the recognition and differentiation of the various types of cardiac irregularity but was of little value in the appraisal of other cardiac abnormalities or in the detection of organic disease of the heart. With increasing clinical use, controlled by experimental and anatomic studies, the value of the electrocardiogram has been more precisely marked out and the scope of its clinical implications has been widened. The electrocardiogram now affords certain criteria of definite value in the diagnosis and prognosis of myocardial disease. Certain types of myocardial degeneration present characteristic changes of the electrocardiogram; other types, less well defined, nevertheless, do afford in the electrocardiogram significant evidence of myocardial disease. Not infrequently, in the confirmation of a clinical diagnosis, based upon history and physical findings, either alone or together, the electrocardiogram is a valuable diagnostic aid. It is often of particular value from the standpoint of prognosis.

This evidence of myocardial disease is afforded by various abnormalities of the ventricular deflections, widening of the R-S interval, notching and slurring of the R-S deflection (which may result in gross deformities of this portion of the electrocardiogram), low-voltage deflections, and various changes of the T wave, such as flattening, inversion, and the so-called diphasic wave. To a certain extent, preponderance of one or the

other ventricle may be recognized from the electrocardiogram. There has been much discussion and study of this point, from which the general conclusion may be drawn that the rules which are usually applied for the determination of preponderance are applicable with certain qualifications as to the build of the patient and the presence of abdominal distention. Of these electrocardiographic changes characteristic of disease, those which are accepted as diagnostic of bundle-branch block are especially typical. This applies almost equally well to arborization block.

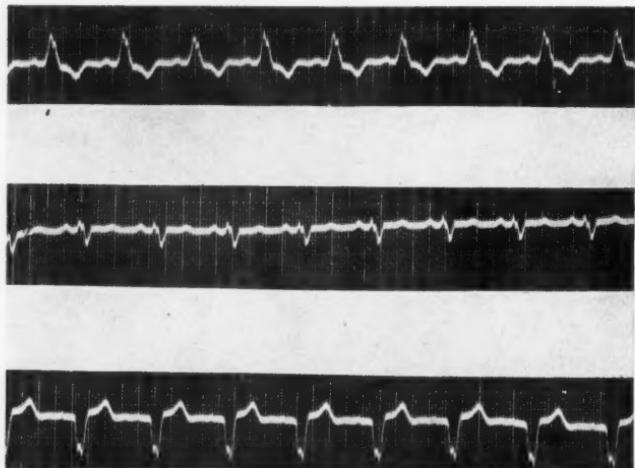


Fig. 15.—Sinus rhythm with right bundle-branch block.

Bundle-branch block is characterized by an R-S interval which is at least 0.1 second in width, by a T wave which is deflected opposite to the main (or R-S) deflection in all leads and by the evidence of either right or left ventricular preponderance. That ventricle preponderates through which the impulse to contraction follows normal paths, hence left ventricular preponderance is found with right bundle-branch block and vice versa. As a clinical event, right bundle-branch block is decidedly the more frequent.

Figure 15 is an electrocardiogram which was of much value in

confirming and in extending our diagnosis and in affording quite definite data for prognosis. The patient from whom it was made was a man of fifty-seven, who stated that for some months prior to this examination he had suffered moderately with shortness of breath upon exertion. About this, he was not greatly concerned. After some months of this dyspnea and about seven weeks before this electrocardiogram was made, he had an attack of paroxysmal dyspnea of great severity. He described the attack as beginning with a cough which rapidly grew worse and was accompanied by a dyspnea which became intense. A physician was called who administered adrenalin which gave prompt relief. Not long after, while on an automobile trip, he had another attack. A physician administered adrenalin at the patient's request and again there was quick relief. For several years he had to control his diet moderately because of a tendency to diabetes. Five years ago, he was under treatment for some months for an ulcer of the stomach. About that time his blood-pressure was discovered to be running about 220. He gets up twice at night to urinate. Physical examination showed a well-nourished, rather obese man, who did not appear sick. The heart was moderately enlarged to the left. The pulse was regular, the rate 68. The blood-pressure was 216/118. There was demonstrable edema of both legs below the knees, and there were a few moist râles at the bases of the lungs. The blood-urea nitrogen was 21.3 mg. per 100 c.c., and the sugar 119. The urine showed a trace of albumin and some finely granular casts. Four twenty-four-hour specimens showed specific gravity in three instances of 1010 and once 1016. The diagnosis of hypertensive heart disease was clear enough; it was also plain that the attacks of dyspnea from which he had suffered were of serious import (incidentally the use of adrenalin was contraindicated in the presence of such hypertension) and likely another time to be succeeded by an attack of angina or of acute edema of the lungs. Yet the electrocardiogram was a surprise to us; the general condition of the man had not led us to expect such pronounced evidence of serious cardiac disease. The electrocardiogram of bundle-branch block signifies an advanced myocardial fibrosis, which

involves one or the other of the main branches of the bundle of His to such an extent as to produce an electrocardiogram like that produced experimentally by ligation of a main branch.

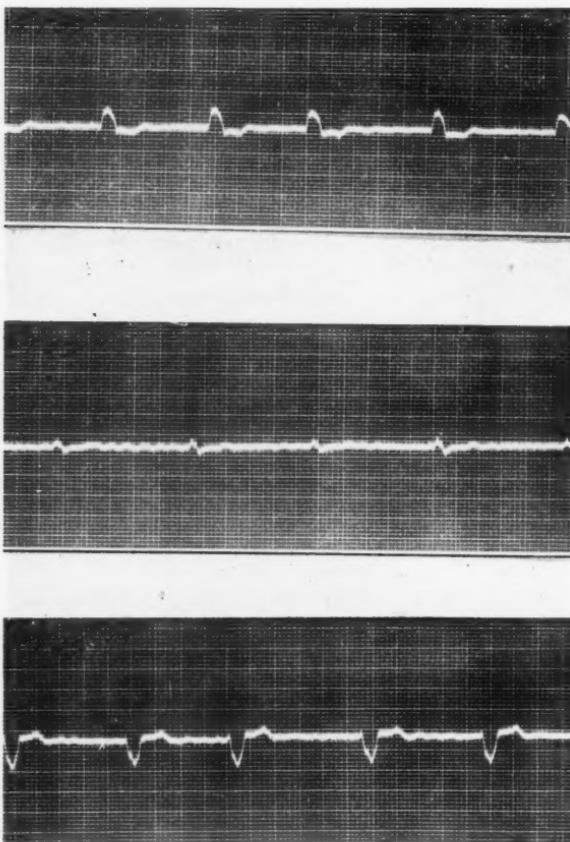


Fig. 16.—Auricular fibrillation with arborization block.

The electrocardiogram of arborization block has practically the same clinical significance. This is also the result of extensive myocardial disease without the typical configuration signifying bundle-branch block. It is associated with marked inhibition

of conduction within the ventricular musculature. The distinction between these two types of electrocardiogram is not always easy to draw; there are, in daily practice, many border-line cases.

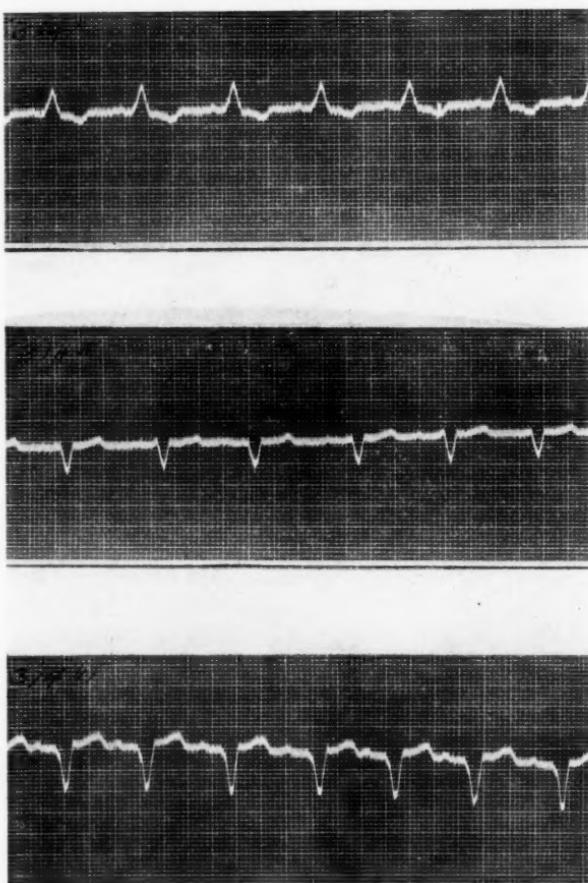


Fig. 17.

But their clinical significance is about the same. Both are due to extensive myofibrosis with impaired conduction within the musculature of the ventricle. Figure 16 is an illustration of arbori-

zation block. This electrocardiogram was made from a man who had been a patient in the hospital several times with decompensation due to myocarditis. The myocarditis was apparently a primary process, without valvular disease or hypertension occurring in a man in the early forties without peripheral evidence of arteriosclerosis. You will note that the T wave is of the diphasic type in Lead I and is deflected in Lead II in the same direction as the R-S complex. The low voltage of the second lead is striking.

Figure 17 presents another illustration of bundle-branch block. This is the electrocardiogram of a man of seventy, who had been operated upon seven weeks previously for hernia. Without undertaking to discuss the indications for operation in this particular case about which we are not informed, it seems worth while to point out that the original record makes no mention of any cardiac disease, neither before the operation nor thereafter. The patient was discharged three weeks subsequent to the operation, apparently in good condition. It had been noted on his chart prior to the surgical procedure that regional examination was negative. There was a statement that some shortness of breath had been present for several years. Following his discharge from the hospital the patient found himself unable to work and had to re-enter the hospital on the medical service. We have no criticism to offer in regard to this particular operation for we know nothing about the particular indications in this case. His distress from the hernia may have warranted the interference even though the electrocardiographic evidence of disease had been known. But the case does serve to emphasize the necessity of conservatism in the employment of surgery in elderly patients and the valuable information which may, at least in some instances, be obtained from the electrocardiogram. Unless the operation was imperative this old man might have been spared not only the surgical procedure, but the subsequent cardiac failure from which he is unlikely to recover.

The significance of notching and slurring of the R-S deflection and of "low-voltage" deflections (abnormally small deviations from the base line) is not easily estimated. These may be

due to errors in technic. The string must be of proper tension, properly standardized, jumping 1 cm. for 1 mv. of resistance and not "overshooting" (it should start from the new

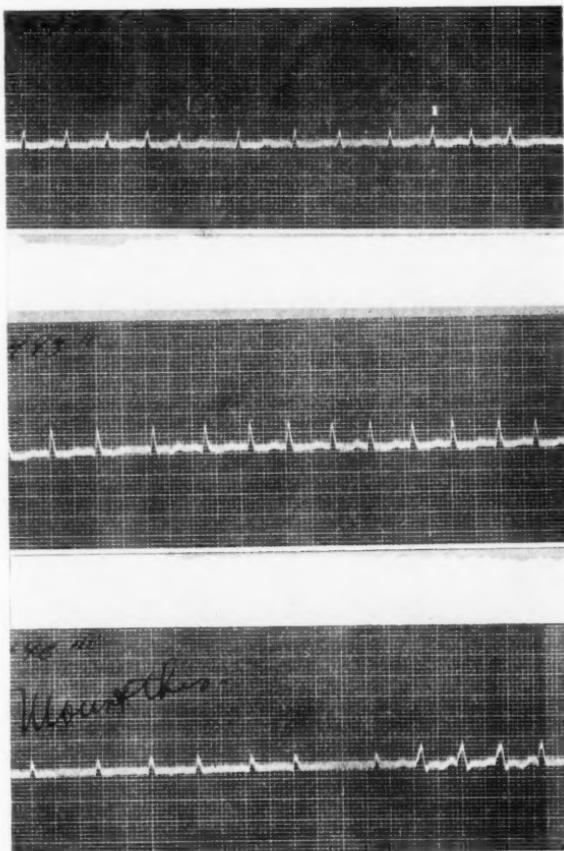


Fig. 18.—Auricular fibrillation: Low voltage in Leads I and III.

level without dropping). With due regard to these factors, the variations in question have significance, not clearly defined, it is true, yet not to be denied. The notching and slurring probably signify delayed conduction within the muscle of the ventricle,

myofibrosis, yet to a lesser degree than is found in arborization block. The low voltage, in my experience, has been apt to be associated with enfeebled hearts, being most often found in older individuals or in patients who have been sick for a long time with cardiac disease. None of these features have significance if confined to Lead III alone; perhaps it is more conservative to say that their significance, when found only in Lead III, is questionable and diagnostic conclusions should not be deduced under these circumstances without careful consideration of all the facts available. Hepburn and Jamieson believe that "low voltage unaccompanied by other cardiac abnormalities is a diagnostic sign of serious import." Burnett and Piltz state that "the occurrence of low voltage in the absence of demonstrable cardiac change, but unassociated with symptoms of limitation of cardiac response, may be considered as extremely suggestive of the presence of cardiac disease." Figure 18 is the electrocardiogram of a man of fifty-five, with arteriosclerotic cardiac disease and decompensation and auricular fibrillation. The voltage is low in Leads I and III, and the R spike is slurred in Lead I and notably so in Lead III. Incidentally, there is a short run (of four beats) of ventricular tachycardia in Lead III; these four beats are ectopic ventricular contractions; the significance of ectopic ventricular contractions in association with auricular fibrillation has been the subject of some discussion and the view that this condition is of bad prognostic import has some support. While it seems likely that the ectopic contractions indicate an unusual irritability of the ventricular muscle dependent upon actual pathologic change, and while some evidence has been set forth to support the theory that the demonstration of ectopic ventricular beats with auricular fibrillation justifies a worse prognosis than when the fibrillation is present alone, this conclusion has not been proved and dogmatic statements are not warranted.

Figure 19 shows an electrocardiogram with a distinct slurring of the R spike in Lead I; this was taken from a young man of twenty-two, who had a history of rheumatism at eighteen, and had been a partial invalid from heart disease for a year. He

had a double mitral lesion. Figure 20 shows slurring and notching of the R spike in Lead I with a broad P wave in Lead II; the latter suggests auricular hypertrophy. The clinical diagnosis

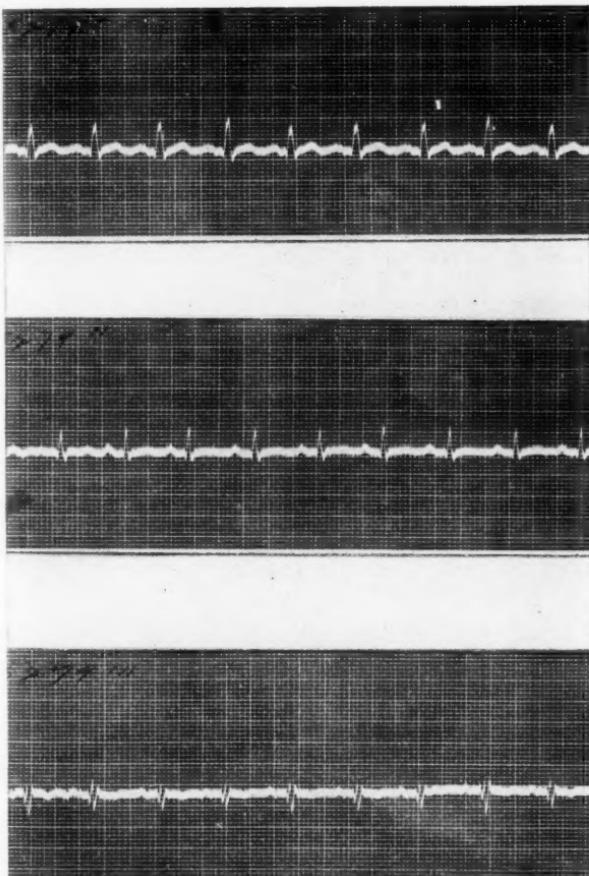


Fig. 19.—Slurring of R spike in Lead I; low voltage in Lead III.

in this case was probable subacute bacterial endocarditis with double mitral lesions and aortic regurgitation; the patient refused to stay in the hospital and was discharged while still run-

ning a fever. Figure 21 illustrates an electrocardiographic change which is strongly suggestive of right ventricular preponderance. While the contour of the complexes and the measurements

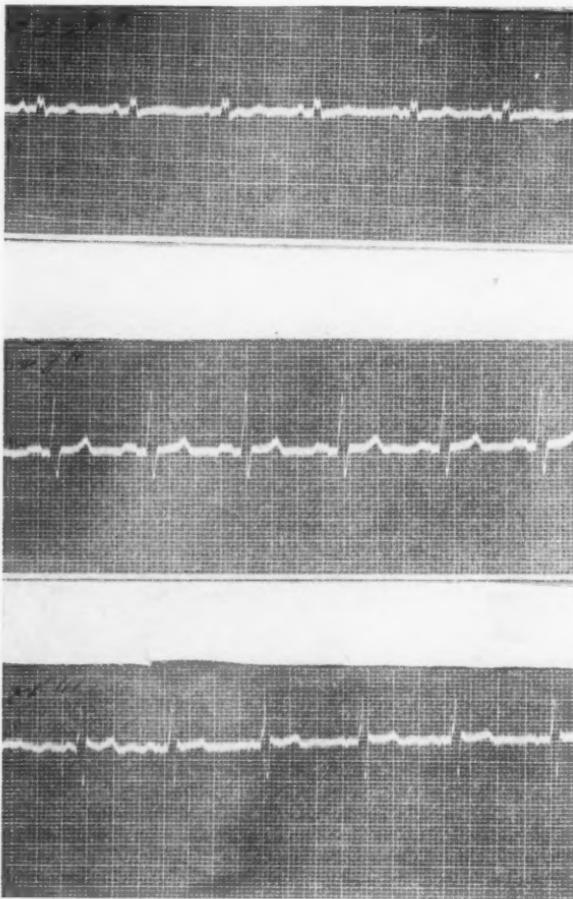


Fig. 20.—Low voltage and slurring in Lead I; broad P wave in Lead II.

thereof do not justify the diagnosis of such preponderance, the high R spike in Lead III in conjunction with the low voltage in Lead I is often found with an enlarged or dilated right heart

such as occurs in mitral disease and the cardiac changes accompanying emphysema of long duration. The R-S complex is slurred in Lead I. Figure 22 is the electrocardiogram of a woman

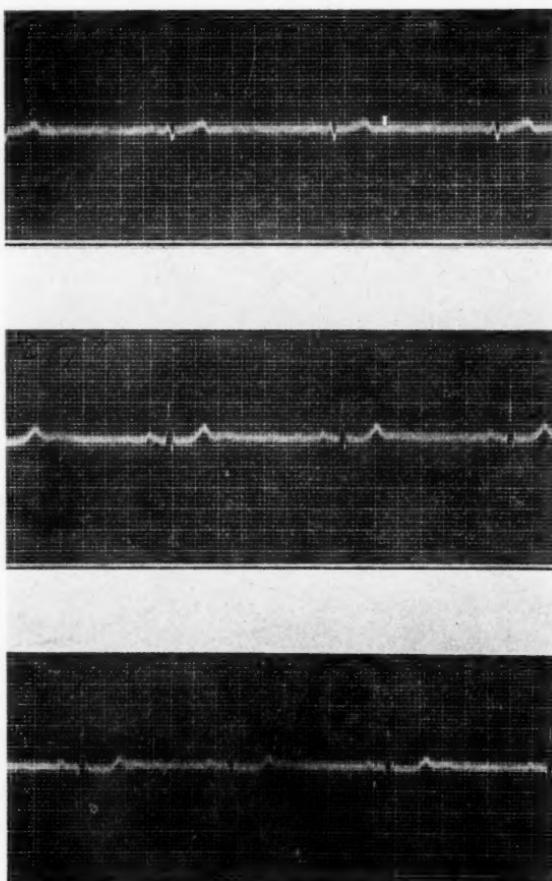


Fig. 21.—Low voltage in Lead I; high R spike in Lead III.

of sixty-two who had a primary carcinoma of the lung. This is a typical "low-voltage" electrocardiogram. At autopsy there was found tumor infiltration of the pericardial sac; there was athero-

matous degeneration of the aorta, and coronary arteries with multiple scars and brown atrophy of the myocardium.

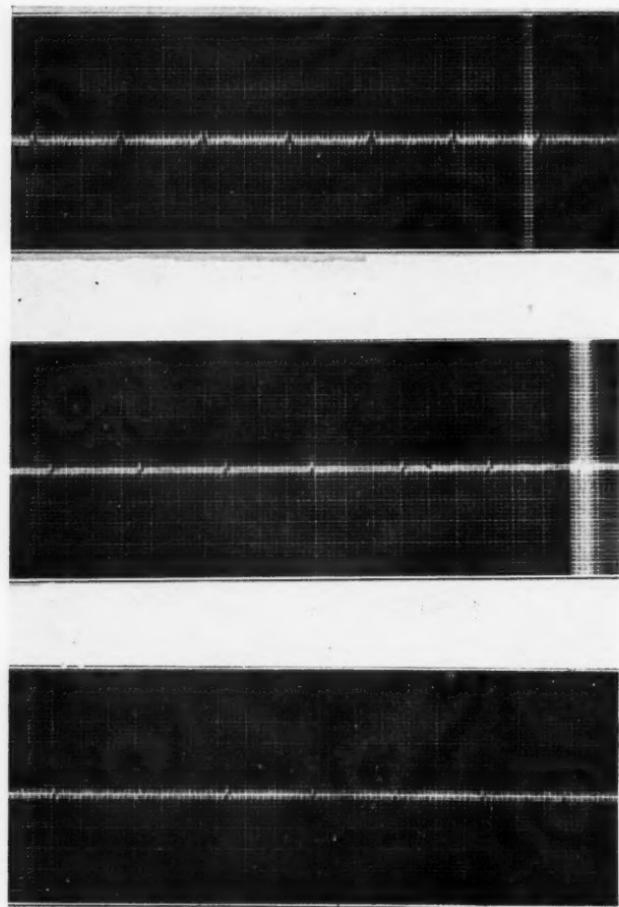


Fig. 22.—Low voltage.

Figure 23 is the electrocardiogram of a man taken some seven weeks after he had suffered an attack of coronary occlusion. At the time of this examination he complained of stenocardiac at-

tacks upon moderate exertion; the physical examination was negative except for a slight enlargement of the heart and weak heart tones. The sharply deflected T wave in Lead I is the most striking electrocardiographic finding, but the R spike is slurred in Lead II and there is left ventricular preponderance. Several months after I saw this patient I learned that he had obtained a life insurance policy for a rather large amount. It is not likely

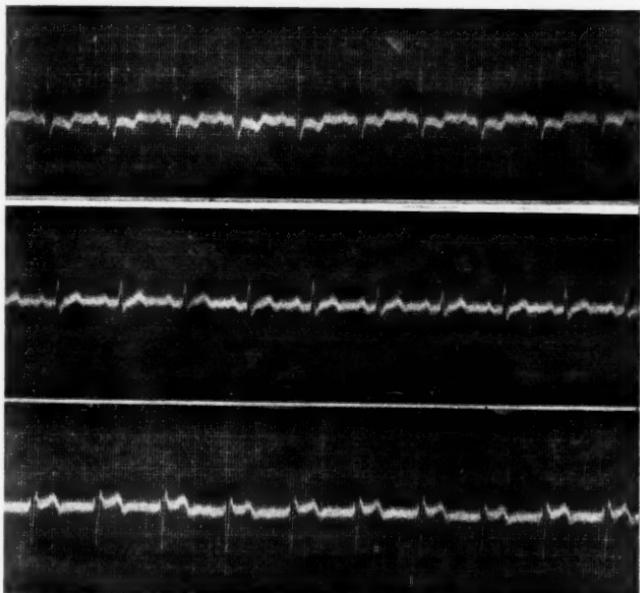


Fig. 23.—Sharply deflected T wave in Lead I; left ventricular preponderance.

that any company would have accepted him if an electrocardiogram had been required before the determination of his insurability.

Figures 24 and 25 are especially interesting. The first was taken from a man of thirty-eight who had suffered for some months with what was taken to be angina, then the electrocardiogram was negative. Later the diagnosis of pylorospasm was made and the electrocardiogram shown in Fig. 24 was made shortly

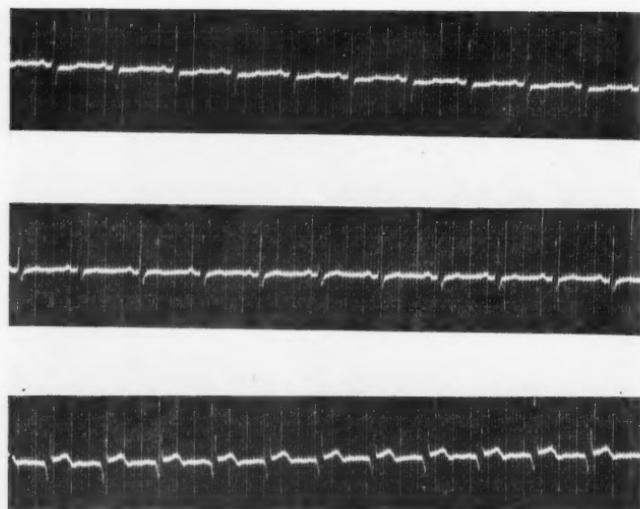


Fig. 24.—T wave, diphasic in Lead I, flattened in II, and suggestive of coronary disease in Lead III.

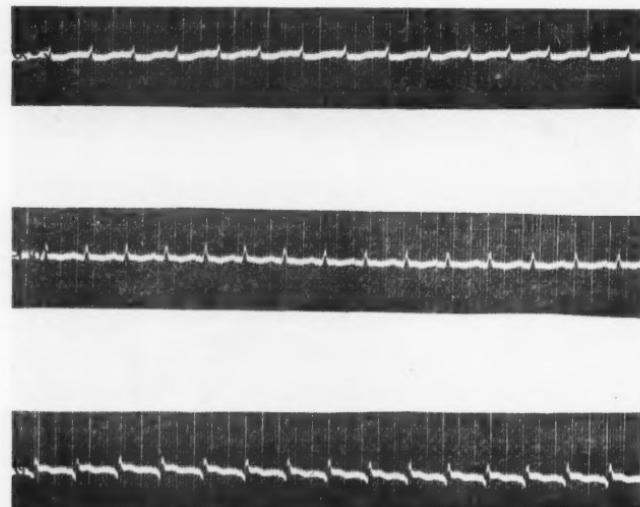


Fig. 25.—Obtained a few days before death from extensive cardiac infarction.

thereafter. The unusual configuration of the T wave in Lead III, resembling the T wave which is seen after experimental ligation of a coronary vessel, again directed attention to the heart. For several months the patient slowly but steadily grew worse, and finally about eight months thereafter he was readmitted to the hospital suffering intensely. There was really no break in the pain during the few days that he lived. The electrocardiogram shown in Fig. 25 was obtained during this time. Autopsy

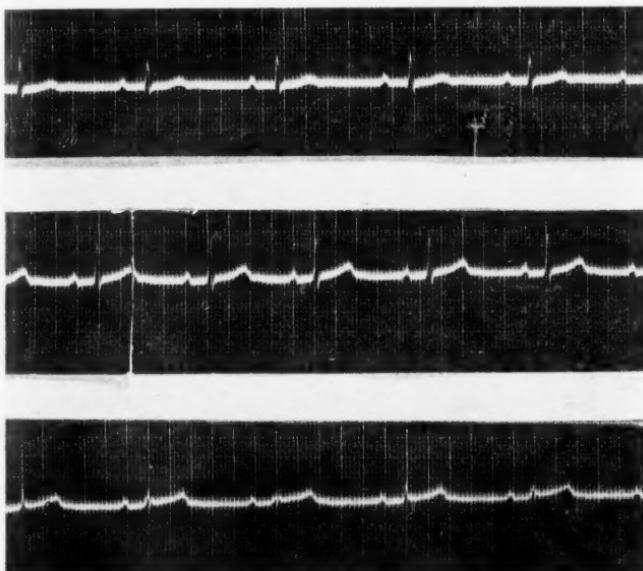


Fig. 26.—Bradycardia: Abnormal T wave in Lead III.

revealed an exceptionally large recent infarction of the left ventricle involving the septum and the apex.

Figure 26 is the electrocardiogram of a boy of sixteen; you will note the resemblance in Lead III to Lead III in Fig. 24. This patient was admitted to the hospital ten days after he had been suddenly taken ill with a choking feeling and shortness of breath. A few days prior to this he had suffered from a sore throat, but had continued to play basket-ball on a high

school team, and it was after a game that he noticed his symptoms. He had noticed some shortness of breath at times for the past eight months, but this was of infrequent occurrence. He

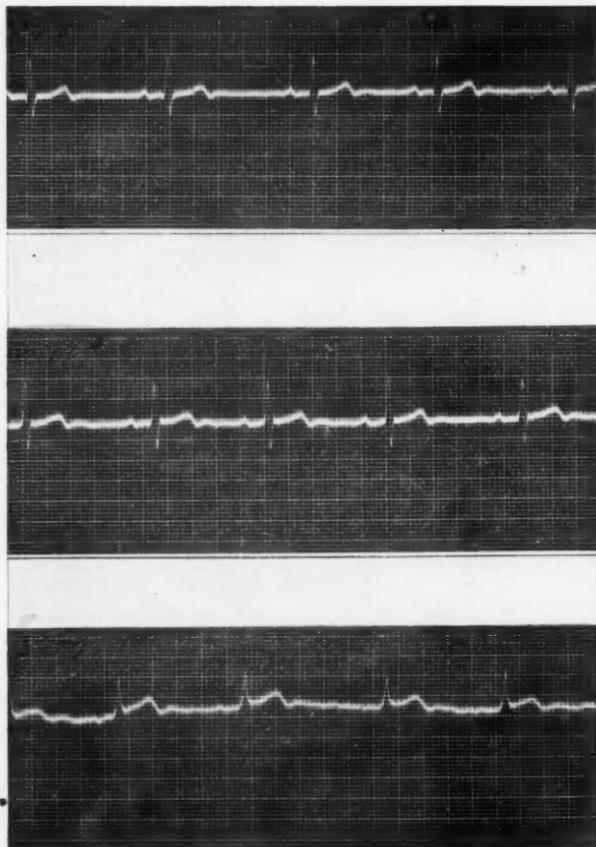


Fig. 27.—Sinus arrhythmia: Compare T wave in Lead III with same in Fig. 28.

gave a history of frequent attacks of tonsillitis. After the onset of this last illness, he had several attacks of choking and through some nights he was unable to lie down at all, but slept in a chair.

Physical examination showed that the left border of the heart was 11 cm. to the left of the midsternal line and the right border 4 cm. to the right of the same line. A loud systolic murmur was

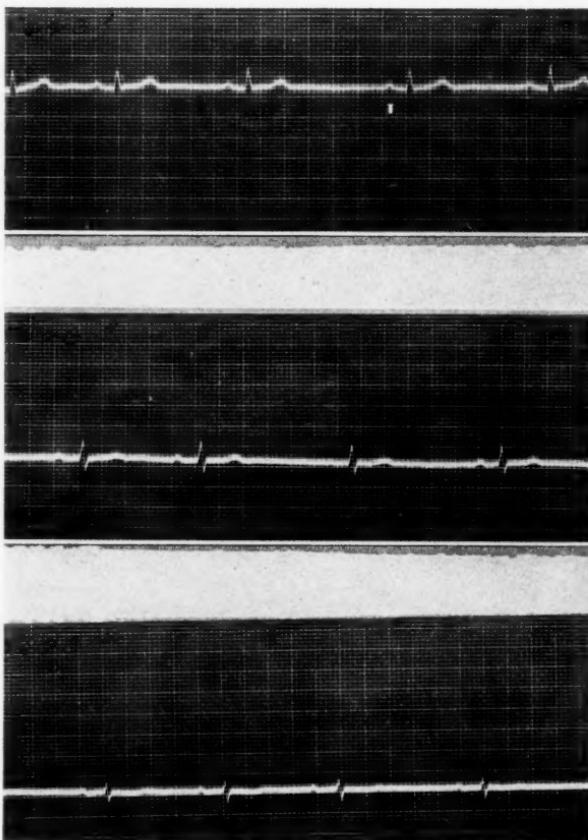


Fig. 28.—Bradycardia sinus arrhythmia: Low voltage in Lead III.

heard at the apex and transmitted to the axilla. The second pulmonic sound was accentuated. The heart rate upon admission to the hospital was 48. He was in the hospital for ten days and rested at home for weeks thereafter. Then he returned to school,

but was kept out of athletics. A year later another electrocardiogram was taken. At this time the patient felt well and

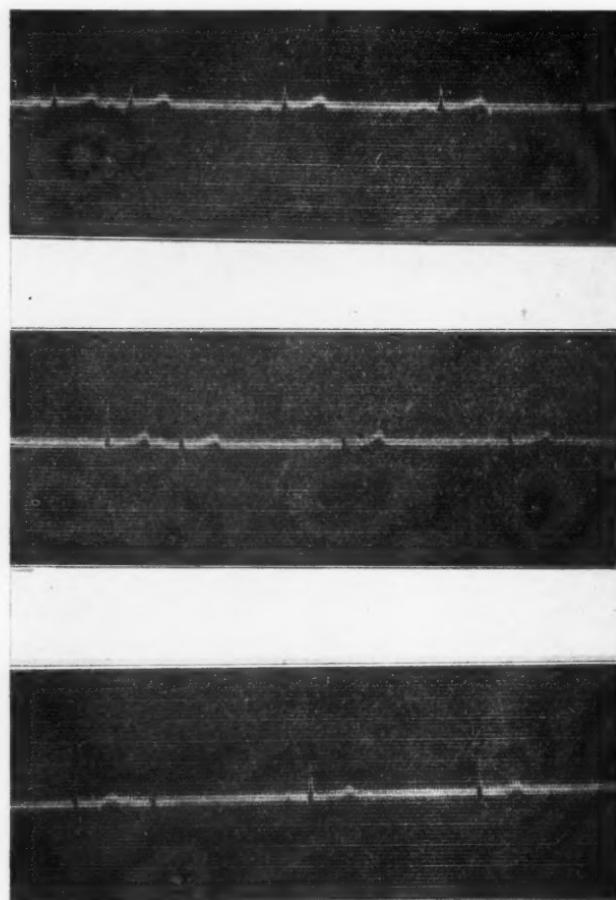


Fig. 29.—Sino-auricular block.

wanted particularly to have permission to take up again his athletic activities. The second electrocardiogram is shown in Fig. 27. The rate is slow (about 54), there is distinct sinus

arhythmia and the peculiar structure of the T wave in the third lead is still present. The significance of this abnormality is not clear, but the existence of localized myocardial damage, such as follows an infarction, is suggested.

Figure 28 was taken from a young woman during an attack of myocarditis after an upper respiratory infection without sore throat. The rate is about 50, and sinus arrhythmia is pronounced.

In general, the complexes are without significance, but the low voltage and the slurring in the third lead would not be ex-

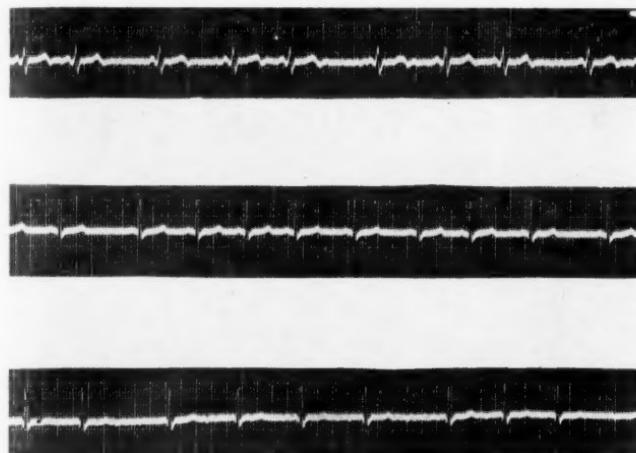


Fig. 30.—Sinus arrhythmia following typhoid.

pected in a healthy young person; these features under the circumstances probably are the result of myocardial change, though, taken by themselves without the clinical signs or history of disease, diagnostic conclusions would not be warranted.

Figure 29 shows the condition known as sino-auricular block. You will note the slow rate with the occasional short intervals, but the predominance of the long pauses. Unfortunately, our technician did not notice the failure of the timer to work. The rate is about 50. This is the electrocardiogram of an old man on whom the diagnosis of senility was made. Figure

30 shows a marked sinus arrhythmia. This occurred during convalescence from typhoid and the irregularity caused a good

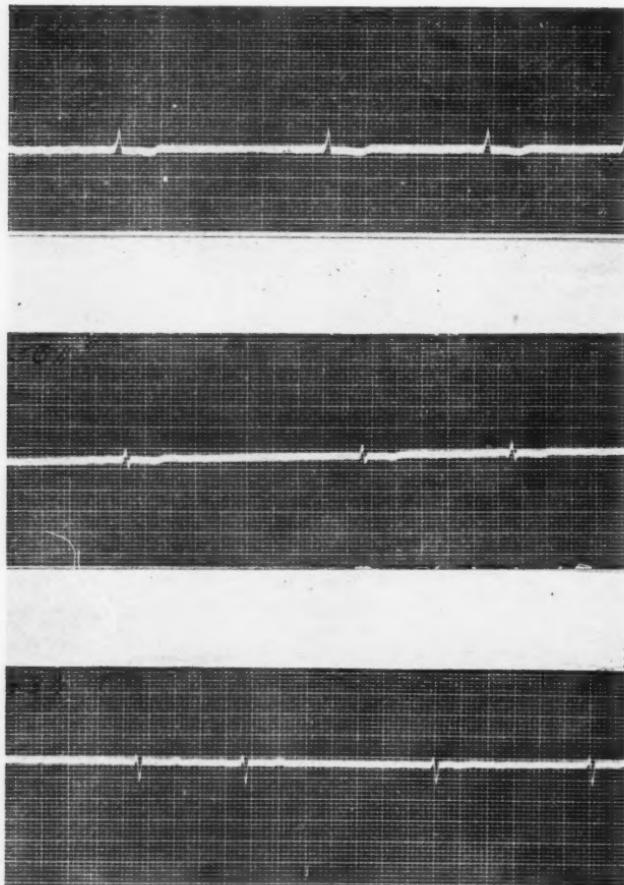


Fig. 31.—Digitalis effect: Auricular fibrillation with slow ventricular rate; depression of T in Leads I and II.

bit of alarm to the family of the patient, who was a boy of thirteen.

Figures 31, 32 show electrocardiograms indicative of an ex-

cessive digitalis effect. The first shows a rate of 40 with the characteristic deflection of the T. As the result of digitalis

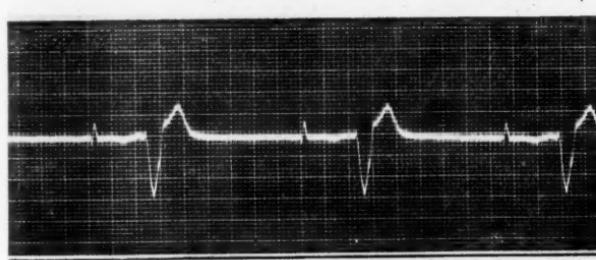
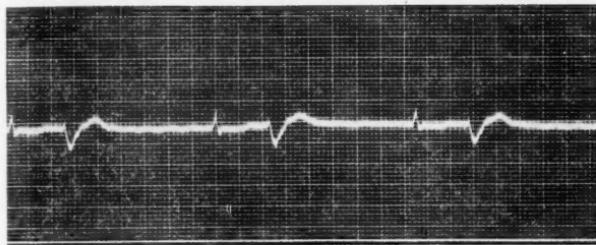
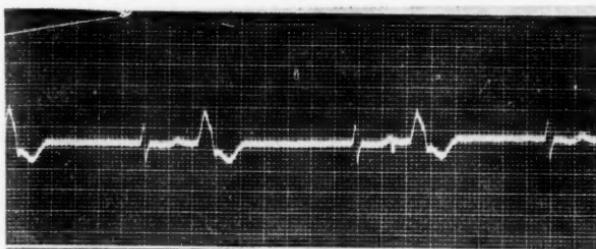


Fig. 32.—Digitalis: Auricular fibrillation with pulsus bigeminus.

the T wave is often depressed, but rarely does this show a sharp deflection. The deflection is almost always of this flat type when due to digitalis. You will note that the rate is irregular, which

means that the drug has produced marked slowing of conduction without complete block. This is an auricular fibrillation in which digitalis has produced marked slowing of the ventricular rate; if complete block were present the assumption of its own rhythm by the ventricle will produce a rhythm which is regular or nearly so. The last figure shows the typical pulsus bigeminus, each normal ventricular complex is followed by one of ectopic origin. Auricular fibrillation is present, as is shown by the absence of P waves and the irregular rhythm.

## CLINIC OF DR. RALPH C. HAMILL

CHILDREN'S MEMORIAL HOSPITAL

### BEHAVIOR DISTURBANCES IN CHILDREN

INADEQUATE or unfortunate adjustments in children are very common. It would seem as though the egoism of children, being young and tender, is easily hurt and can be counted upon to make its hurts known.

A child knows what it wants to do: It wants to hear the crash of the falling china. That the china is mother's best and will be destroyed makes no difference; mother is that person who is always saying, "Don't," and children, lacking a sense of continuity and always finding entertainment in the new and so not missing the old, have no regretful feelings about that which is destroyed. To hear the crash is exciting, and so the table is pushed over.

When the wreck is discovered the offender is punished. Thereby an attempt is made to teach him that his wishes must take account of the wishes of others. We may say that his ego has to fit into a certain pattern; has to adjust to a social convention. The resentful crying that follows the punishment is convincing evidence that the adjustment is painful. (All of us hate to be hurt, especially in our feelings.)

Punishment is supposed to teach the offender that his offending will bring him more pain than pleasure. Maybe it does, but it also arouses a resentful type of anger in a great many instances. This may be thought of as the reaction of the ego to its forced adjustment.

Resentment and anger show themselves in many ways, and where they go up against a degree of superiority upon which they can make no impression, fear is the result. Mathematically it might be expressed thus: Resentment and anger plus impo-

tence and discouragement give fear. This formula covers the emotional elements of maladjustment, behavior disturbance, mental disease, the psychoneuroses, and the psychoses. These emotions lead to action, but they seldom lead to straight thinking.

Behavior difficulties of children are of the same type of thing as psychotic or insane behavior of adults. Both are attempts at instinctive expression, not trimmed and molded to the conventional forms. The essential difference lies in the field of the imagination: The bad boy of the school age misbehaves to get the laugh of the others (their attention); the paranoid precox patient knows that everyone is looking at him. The former does something to get attention complimentary to his ego; the latter has been able to convince himself that he has that attention, that his ego is so important that all the world is watching him. The trouble is that both are allowing their egoism free rein at the expense of others instead of adjusting themselves to others.

It seems as though there are two important elements behind all behavior difficulties: Fear and rebellion.

Why children should be fearful and rebellious is a question. We try to answer it, but a thorough-going belief in any explanation is far from common property. It may be true that every boy loves his mother and every girl her father, and so they hate and fear the marital partner. It may also be true that there is a drive for freedom and independence inherent in the flesh; of the fetus that grows too large for its mother's body, of the boy who breaks the apron strings, of the girl intolerant of the boarding school, of the adventurer and explorer. It may be that the gospel of self-control makes children intolerant of the control of others. At any rate, back of the behavior difficulties described in the following cases fear and rebellion loom ever present.

There are many ways in which a child shows that it is in difficulties. To cry and to show fright are universal. The cry may be of pain, physical or mental. It may also be of rage, resentment, and rebellion; for sympathy or attention. Children are soon exposed to the idea that crying is not an acceptable method of self-expression, and yet they go on doing it. Tell a

child something it must not do and you increase the chances of its being done. And so all the activities that can be covered by the general heading of "mischief" are to be included.

Enuresis is a failure or unwillingness to subscribe to the standards of adults. Night terrors and tantrums are crude fear and rebellion. They come in bursts as attacks, out of apparent normality, and are followed by a return to normality. In some cases of tantrums breath is held to cyanosis, the child gets rigid and fails or refuses to respond, as though unconscious. Descriptions of the early attacks of some epilepsies are much more suggestive of tantrums than of anything else. There is a variety of ways in which children show resentment against competition. They may offer to kill baby brother or sister, or merely refuse food that has been pushed into their mouths when they know that the baby is enjoying its mother's breast. If a child tires of the competition at school it may develop headaches, dizzy spells, coughs, tics, vomiting spells, or any other physical complaint in order to avoid return to school.

In these cases fear and rebellion lead to a great range of unfortunate activities. There are restlessness and inattention, poor sleep and night terrors, enuresis and self-soiling, tantrums and fits of breath holding, capricious appetites and vomiting, headaches, and so forth.

Drugs are not indicated, nor are operations. Removal of tonsils and circumcision *can* be more harmful than beneficial. Patience with parent and child are absolutely essential. Enuresis is not to be cured by dry diets and belladonna, nor restlessness and terrors by bromid. The elements of fear and rebellion must be sought out and dealt with directly.

As a general rule it may be said that the younger a child, the simpler is the problem, the more direct the expression of rebellion.

Attacks of rage with associated breath holding are such direct expressions.

**Case I.—**J. P., aged three years, one month, a chubby, rosy-cheeked youngster with large, wide-set blue eyes, who looked more like an overgrown kewpie than anything else, was brought to the clinic by her mother because

of holding her breath and turning blue when she got angry. She had done this since one year of age. She would grind her teeth and seem weak after these attacks. Three months before she came to the clinic she began to fall over and become unconscious for two or three minutes, get stiff, clench her fists, roll her eyes upward, and at times her arms and legs would draw up. After these attacks she would cry and want to go to sleep, but her mother would not let her for an hour or more; then she would go to sleep for several hours. These attacks came about three times a week and seemed to be getting worse. The child would always urinate in the attack. She was easily frightened and particularly afraid of a dog, an automobile, and Santa Claus. She had a quick temper, held her breath when angry, and was then cross and sulky. She was active, affectionate, and liked to be noticed. Her feelings were easily hurt, and whenever they were, she would pout for a long time. She did not get along with her older brother, she was jealous of him and wanted whatever he had. She played with other children who would baby her. She always seemed happy when getting the attention of everyone around her.

The mother, aged twenty-two, was married when she was sixteen. She had been "nervous," and at thirteen it was thought for a time that she had chorea. She went one year to high school and then was an addressograph operator until her marriage. She stated that she had diabetes, but could not afford treatment. She seemed co-operative and, although skeptical, was willing to try the doctor's recommendations.

The father, aged twenty-two, was a helper on an ice wagon. He was in his second year of high school when he married, at seventeen. He was very fond of Janice, but saw her only on Sundays. A brother, Francis, aged five, seemed to be the mother's favorite. He teased the patient a great deal.

Physical and neurologic examinations were negative. At first she seemed afraid of the examiner, but later made friends with him. The mother was told to leave her alone when she started having a spell and not to go back to her until she was over it. Two weeks later, when the mother brought the child to clinic, she stated that she had had only two spells in those two weeks; the same method of treatment was advised, and the mother was to leave the patient with her grandmother as much as possible, since the child had never had a spell when with her grandmother. Six weeks later the patient had had only one spell. This one had been at her grandmother's when it was time for her to go home and the grandfather wanted to wash her face. At this time the grandmother carried out the doctor's recommendations and the child was left alone. When the seizure was over the grandfather took her home.

The mother continued the doctor's recommendations, and

during the last three and a half months Janice has not had a seizure. She is still jealous of her brother, wanting whatever he has. The mother is attempting to get Janice to play with children of her own age. She preferred to play by herself, but after she got to playing with other children her improvement was immediate.

It was explained to the mother, with Janice in the room, just what the cause or purpose of these spells was. The mother was told that because the spells were a demand for her attention she was to let Janice alone, let her get over them by herself. It worked.

**Case II.**—Margaret K., aged five years, seven months, was brought to the clinic by her mother because she was living "in terror" day and night. Almost every hour during the day she would come running to her mother crying, "Oh, mama, look, there it is again, take off my clothes." The mother would take off her clothes and show her there was nothing wrong, but Margaret would stand trembling with her hands on her hips, and apparently afraid something was going to harm her in the genital region. At night she would not go to bed without her mother, and the latter would have to sit by her bed until she went to sleep. She would not allow herself to be covered from the waist down, would not even tolerate a nightgown. After she had gone to sleep the covers would be put over her lightly. If she wakened and found them over her she would "almost go crazy." The light had to be left on, and at times she would waken screaming so loudly that the neighbors were aroused. She usually wakened about 3.30 A. M., and her mother would have to sit up with her for the remainder of the night.

The mother, aged forty, was a sensible, co-operative Irish woman, who spoke with a delightful brogue. She was tired and discouraged when she brought Margaret to the clinic, as the child had been carrying on this way for six months. The father, aged forty-four, Irish, was extremely fond of Margaret and she of him, but when she began her spells she would have nothing to do with him. A sister, aged sixteen, seemed to be the favorite of the child, and this sister was inclined to spoil her.

Physical examination was negative. Psychometric examination gave an I. Q. of 100. The psychologist found her to be a normal child in every way, eager, attentive, and friendly. When she came to the psychiatric clinic she denied being frightened, and showed a ready smile when anyone smiled at her. She cried very affectedly when the mother started to leave her alone with the doctor. Because of her crying no further examination could

be made at that time. On her next visit to the clinic, two days later, the mother reported that the child's panties were unbuttoned and that she would not permit them to be buttoned. Margaret cried when this subject was mentioned. She had continued having the terror spells. Because of her continued crying and because of inability to get her to talk, except to deny each thing in turn, she was placed in the hospital.

When she realized this she cried, held her breath, danced up and down, fought at worker, trembled all over, and clung to her mother. Her screams could have been heard a block. Finally, when she saw this was not having any effect she began to beg, "Please mummy, take me home, I'll sleep all night," "Please mummy, don't leave me here, I'll be good."

The first night in the hospital she caused much trouble, would not sleep alone, and demanded the light. The second night was a little better, and the fourth she slept the night through. After eight days in the hospital the first night at home was as disturbed as before she came to the hospital. After the mother had gotten up a few times she told Margaret she was not going to get up again, the doctor had told her not to, and she was going to carry out his instructions; Margaret could cry if she wanted to. After a few nights Margaret began sleeping the night through. In clinic she said she had been scared by a dream, but could not, or would, not, remember it. She still would not permit her mother to leave the room, had another temper tantrum. On the following visit the mother left the room in spite of Margaret's crying. It was explained to her that she could not have her mother with her always. She quieted down, but continually asked if she could go home with her mother. At her next visit she went in alone to see the doctor. She had been put in kindergarten, was going every day, and not causing any trouble at home. Whenever they would not let her have her way, instead of a temper tantrum she would say, "Well, the doctor said I had to obey you, so I suppose I will have to." This would be said with a smile.

Margaret told the doctor of going to the zoo, where she saw a monkey. Her older sister told her the monkey would get her.

Also, that there might be things in her bed. She was asked if she was afraid that something might crawl into her. "Yes, it would crawl way through me," said with considerable animation. "Where would it crawl in?" "My mouth," she answered in a much less naïve manner. It was obvious that the answer was an evasion, since, of course, her mouth was not under the covers nor under her clothes.

Now, three months have gone by, she is very friendly with the doctor, and the mother says that life is worth living, Margaret is a changed girl.

**Case III.**—Margaret D., aged eleven years, ten months, was referred by the parental school because she was never still, did not do her work well, and kept the other children upset. She and her sister fought a great deal. It was necessary to put Margaret in a private room some distance from the dormitories. The two sisters had been placed in a parental school for stealing, truanting, and misbehaving at school. At clinic Margaret had a hard-boiled attitude, was dilatory, and answered "Don't know" before she considered a question. She seemed indifferent, but when a little attention was shown responded quickly. The parental school reported poor sleep, restlessness and screaming in the night, spilling everything on her clothes, seeming not to find her mouth when taking food to it. Before the parental school she had attended five other schools, two parochial. In the last two schools attended she was repeatedly in the principal's office and up before the truant officer.

The mother, aged thirty-seven, a Bohemian, seemed incapable of managing her girls. "There is nothing the matter with them, but everyone else is picking on them." She worked in a box factory, earning \$17.00 a week. The father, Michael, aged thirty-seven, Irish, a drunkard, had deserted his family two years before. He never supported them, drank, gambled, and was frequently abusive.

Physical examination was negative. Psychiatric examination showed her restlessness, indifference to questions, complete lack of confidence in the examiner. In school Margaret would frequently hide in the toilet, light the gas heater, and set papers on fire. At one time she tried to set fire to a little girl's clothes. She was the leader of "the gang," which was known as the "Secret Ambition." Apparently the children were hiding in

toilets to see under other children's clothes, both boys and girls. Much of their conversation was about babies, and the relations between men and women. This gang practised mutual masturbation. The symbol of the club was a key drawn on a piece of toilet paper. As one member came into the toilet she was given the symbol and kept it only until the next member came in. On Margaret's return to clinic masturbation was discussed with her. She admitted the activities of the gang, as described. A week later she told of an episode with six boys. Two weeks later the problems of sex were again discussed, and the teacher reported that she was causing no further trouble in school.

Six months have elapsed since this last visit to the clinic. She has caused no more trouble, has settled down in her school work, and in the last months has been permitted to return to her home. She is again going to parochial school and the Sisters are keeping her until late in the afternoon.

**Case IV.**—Donald P., aged six, was referred by the medical clinic because his mother said he did not advance mentally, could not reason, said irrelevant things, did not play with other boys, and became excited easily. He masturbated occasionally, and wet his bed. He was energetic, lively and talkative, saying the same things over and over. When anyone called at the home he would get excited and race from room to room. He occasionally had dreams which frightened him.

The family consisted of the father, aged thirty-five, a clerk for an ice company, a World War veteran, getting a small compensation. His feet swelled and hurt badly. He frequently came home cross, irritable, and did not want the children around. The mother, aged thirty, was very precise and particular. She felt her surroundings and the lack of luxuries very keenly. There were two little sisters, normal. The maternal grandmother also lived in the home.

Physical examination showed tonsillitis and bronchitis. It was impossible to establish a fair intelligence quotient. The patient was very nervous and gave answers that had no bearing on the questions. When I examined the boy his mother constantly interfered, telling him what to do. He stammered when he first entered the room, but later spoke fairly well. He stated that he was afraid of noises, thunder, "cluck," and barks. The mother was advised to put him in overalls, let him get dirty,

play in the school playground, and that she must stop correcting him, give him constructive toys, let him alone, and not call attention to his timidity.

Six weeks later a social worker called at the home, and saw Donald playing in the playground much improved. Shortly after this his tonsils were removed. A month later the mother reported that when the patient was left alone he was normal and communicative, but self-conscious when interfered with. She was again advised to stop criticising him, to let him play with other children, and put him in school. Two months later the enuresis had stopped, he was doing well in school, recited several of his school pieces, gave sensible answers to questions, and seemed entirely different than on his first visit. Also, he was playing nicely with other boys.

A psychometric examination showed an I. Q. of 88. The apparent dulness of the first visit had entirely passed out of the picture.

**Case V.**—Donald L., aged six years, two months, was referred from the medical clinic, suspected of chorea. He was hyperactive, and had had night terrors for the past six weeks. During the day he would shake his shoulders and pull at his clothes. In school he was fidgety, cross and irritable, sensitive, fussy about his clothes, whined, and cried. He was babyish and liked to be petted.

The family consisted of the mother, aged twenty-five, who was nervous and "taking a tonic." The father, aged twenty-nine, was strict but fair in his discipline. There were two boys younger. The maternal grandmother was also in the home. She was nervous, cross with the children, taking care of them from time to time when the mother worked.

Physical examination was negative. Psychometric examination gave an I. Q. of 89. The psychologist said he was slow in reaction, required much urging, restless, bit his nails, and walked the floor. During the examination he made references to animals that jumped on men and killed them, and would say immediately afterward that he wanted to go to his mother.

To me he stated that he waked during the night and would dream "It ate my brother, and when he made him again he had a long head, then it made him a little head." I explained to him that he wanted to get rid of his little brother. The night

terrors were discussed as things he could control. The mother was told to leave him in his own bed and pay no attention to the bad dreams. Of course he was present as this was said. On his next visit to clinic there had been no further night terrors. Three months later the mother stated that he was entirely well.

**Case VI.**—Irving K., aged seven years, two months, had been treated in the cardiac clinic for chorea, which developed after he entered the first grade of school. After a long rest in bed he improved, and at the beginning of the next term he returned to school. He was sent home, however, a few months later, because he was fidgety, walked around his seat, and fought with the other children. She thought that the teacher was too severe with him. When the other children called him "Dumb bell" and "Baby," it angered him, provoked fights, the teacher blamed him, made him tremble and lose control of himself.

The parents were Jewish and very excitable. There was an older sister who bossed him and gained his resentment, a younger one was a good playfellow.

The psychometric examination gave an I. Q. of 92. The boy seemed extremely slow and, at first, gave the impression of dulness.

A visit to the school revealed the fact that Irving was sent home because he soiled himself. This made him objectionable to the teacher, and the butt of the other children's jokes.

I talked with Irving about soiling, and pointed out that other children would continue to call him names as long as he continued the habit and probably for quite a time after he stopped it, in fact, until they forgot about it. He was given to understand that he would stop if he really wanted to, and that if he wanted the respect of other children he must stop. Soiling was a baby habit, and he must be a regular fellow.

He came to clinic several times thereafter, each time reporting no soiling. He still seemed very timid, and it was discovered that he was very much afraid of policemen. Policemen were discussed with him as boys' real friends.

The last time Irving came to clinic he reported that he had stopped soiling and that the children had stopped calling him names. He had been promoted in school and was not nervous any more. When asked if he was still afraid of policemen, he

said, very slowly, "No." I discussed policemen again, frontward and backward, and Irving went away laughing at the idea that he might some day be the chief of police.

**Case VII.**—John H., aged seven years, ten months, was referred from the medical clinic because of walking and talking in his sleep. One night he unlocked the back door and called, "Goodbye, mother, I am going away." Another night he urinated under the kitchen sink and then went to the bathroom and flushed the toilet, and so to bed. He frequently looked at the clock while walking in his sleep, saying, "I haven't much more time, have I?" In the morning he would tell his mother that he had had terrible dreams.

He was very fussy, away from home wiping his plate and silver with the napkin, and if the silver was worn he would say, "I can't stand to eat with it," and would not. At home if his uncle had been in John's chair, John would refuse to use it until it had been scrubbed. He seldom went to a restaurant or soda fountain because he was afraid the dishes would not be clean. He would not go into stores because of the odor. This condition had been getting progressively worse for about a year. At one time he became so excited that his mother put him to bed, where he remained for two days, and then said he would go crazy if she did not take him up. He could not stand this kind of shoe, or that kind. His mother tried different kinds of underwear, but he always complained. He complained of the baby: she made him nervous, he could not stand her crying. If she accidentally touched his chair he would almost have a spasm. In school he would ask the teacher to make the other children stop making so much noise, "he couldn't stand it." When he did not get his way at home he would threaten to pack up and leave. He wet himself, twitched his face, and stammered.

His father was easy-going, his mother very efficient, but an extremely exacting woman, easily irritated, who said that John made her so nervous at times that she could not stand it. His sister was five years old.

Physical examination was negative. Psychometric examination gave an I. Q. of 120. It was explained to the mother that John's reason for finding fault with everything was because he had been found fault with so much. She was advised not to call attention to his weaknesses, and to forget cleanliness and neatness.

John and his mother have been coming to clinic for six months. It has been extremely difficult to get her to relax her discipline, but she has tried. After a time she put John in overalls and allowed him to acquire a little dirt.

John's pride was appealed to, with good success. Three weeks after the first clinic visit enuresis and sleep walking stopped.

Before going to bed at night he would say, "I am not going to do any of those things." He ceased to wipe his plate and silver and made complaints only very rarely.

There was a relapse, however, at school. The principal had not promoted him as recommended. He became lazy and his grades slumped to 50. He said the girl behind him bothered him. The teacher demoted him, and his mother brought him to clinic because he was restless and worried about failing. I encouraged him, and later found that he had been restored to his old class.

Two weeks ago he reported that he had been again promoted, was enthusiastic about his new work, had stopped his misbehavior.

**Case VIII.**—Robert J., aged ten years, six months, was referred to clinic, unable to get along in school and everyone picked on him. He had been in every other clinic in the dispensary since the age of three months.

The father, aged forty-four, had been deaf and dumb since one and a half years of age. He was not successful in business and was frequently out of work. When Robert was one year old he deserted his wife and child; six months later he returned. The mother, aged forty-two, was also a deaf mute. When I first saw her she and Robert both seemed frightened and very ill at ease. However, she was rather accusatory and belligerent, she seemed to demand rather than appeal; also she impressed me as though she was accusing the world of mistreating her.

The boy, though somewhat confused, was also on the defensive, and it was soon obvious that he told his mother only what he wanted her to hear, talking to her in pantomime and sign language, saying a great deal with only a few gestures. Before saying anything to her he would pause several seconds, thinking just what he was going to say. Then he said only what was calculated to preserve a certain image of himself and his relation to the outer world in his mother's mind. That image was that of a small, defenseless boy, unfortunate in his parents, unjustly treated at school and by the world in general, and deserving constant, tender consideration by his mother. To the end that this image be preserved he had convinced his mother of the meanness of the children at school. He was picked on by everyone, and partly because his parents were deaf and dumb he had troubles, and it was her fault.

His mother, in turn, was trying to shift the weight on to the kids at school, and "Why didn't the teacher do her duty and see that my boy was protected?" "Why didn't she make them behave?"

In school Robert did poor work, and was absent from six to fourteen days a month, complaining of headaches, nausea, pain in his stomach, and so forth. His teacher said he was lazy, shiftless, tormented the other children, and was always in difficulty. He was a tattle-tale and blamed others for everything that happened. His principal, who had known him for several years, said he was the meanest boy to his mother that she had ever seen.

Physical examination was negative. Psychometric examination showed an I. Q. of 80, with particular deficiency in vocabulary and language tests.

To me he seemed confused and on the defensive, talking as little as possible. It was always the mother who told how mean the children were to him, the patient saying very little about it.

The belligerent, accusatory attitude of the mother antagonized the teacher and the neighbors, instead of helping the boy.

The outlook in this case at the end of the study was not cheerful, but it was decided to carry it along as an intensive treatment case. The medical recommendations were that he have a tonsillectomy and adenectomy. Psychiatric: Intensive treatment to assist the patient to adjust himself to his difficulties, and to train him in better methods of adaptation. Educational: The patient should not be pushed in school, as his mentally inferior ability was not such as to warrant it; special attention should be given to vocabulary and language. Social: Intensive work with the mother to effect a change in her attitude, get her to adopt a more grown-up attitude toward the patient, to get him more interested in being grown up than in remaining a baby, and to stop believing all the petty little things he told her. Intensive work with the father to get him to take more interest in the patient by taking him to various places of amusement, and to try to learn to understand him better. To attempt to get the mother to send the patient away for the summer, the building up of social contacts for the patient's encouragement, play activities with the neighborhood children, placement in a boys' club, gymnasium, and social activities.

An improvement in the mother's attitude was obvious just as soon as we worked directly with her instead of through the patient. Robert's attempts to present a certain picture of his difficulties were explained. She seemed to understand this and to appreciate that she and the father were ruining him by keeping him a baby. Frequent home visits, when the mother and father were seen together, were necessary. Getting the different points over to them was extremely difficult because everything had to be written out.

The first step in this change was to get Robert to sleep alone. She soon stopped going to school and causing a fuss. She stopped listening to his little difficulties, and encouraged him to fight his own battles if this was necessary. He was permitted to join the Boys Club, where the head worker took particular interest in him and served as a man ideal for the patient. He was sent to camp for six weeks during the summer, where he fitted into the camp life and enjoyed it so much that he wants to go for the entire summer next time.

This case has been under treatment for nine months. The mother has entirely changed; she is friendly, co-operative, willing to carry out any suggestions of the clinic, and quite happy in Robert's ability to get along. He has been to the clinic only once in six months, and this was to have a cut on his face dressed. The cut was the result of a fight in which Robert stood his own ground. The mother was rather amused at this, not at all worried, and did not even scold him for getting into a fight. The social worker continues to see her occasionally and the mother is keeping up her attitude with Robert. The father also is quite different, and it seems that they have found someone who is not down on them because of their deaf mutism. This has apparently made a complete change in their attitude, as well as in that of the patient.

**Case IX.**—Vincent L., aged eleven years, three months, had been known in the dispensary since he was five years old. At that time he was brought in for being finicky about his food and being "quite nervous." His tonsils and a few glands in his neck were moderately enlarged. He was brought in three months later because he was subject to vomiting spells. The mother was given disciplinary advice and told to return. Next the boy was treated in the eye clinic, and later for a pus infection in both legs. The next year he was brought in because he was under weight. He was put on a diet and nothing further was heard of him for two years. The mother then brought him in because he was having trouble in school, was mischievous, and played truant frequently. At this time he was referred to the psychiatric clinic. The mother stated that he could not sit still, was fidgetty, noisy, and made himself a general nuisance in the house. He could not get along with other boys, she could not believe anything he told her, could not trust him, and he would not obey. Whenever he was punished he would get into a temper, threaten to jump a freight train and leave home. He would tease, was defiant, fought a great deal, and was described as being aggravating in every way.

A visit to the school revealed the fact that he was annoying to his teacher because he did not sit still, because he was constantly in difficulty with the other boys, and because he was always telling on the other children. His teacher was about ready to send him to the parental school. The principal had quite firmly decided that he masturbated and that this was the cause of all his trouble. In spite of this, however, Vincent was up to his grade and had never failed to be promoted.

The father, aged thirty-seven, German, had been in this country for fourteen years. He had been through the grade schools in Germany, and had worked as a barber since coming to this country. He had never cared for the patient and did not want him, being quite mean and antagonistic to his wife when she was pregnant. He seemed to resent having to support his family, preferring to spend all he made on himself. He would never take any notice of Thanksgiving, Christmas, or birthdays, saying they were no different than other days. The mother, aged thirty-four, was born in England. She went to grade school, then to business school, and worked as a stenographer until after she became pregnant with the patient. She seemed to be a sincere, hard-working woman, but given to complaining. She was co-operative, answered questions freely, and seemed anxious to carry out the doctor's advice. A sister, aged seven, completed the family group. She was the father's favorite, and everything she did was perfect in the eyes of both parents. The family history was negative, except that the maternal grandmother lost her memory and was "queer" for several years prior to her death. The maternal grandfather died from tuberculosis.

Physical examination showed a well-developed boy, slightly under weight for his height. No neurologic examination was made. Psychometric examination gave an I. Q. of 100. His problem seemed to be one of application. At first he was suspicious of the examiner, and later rebellious, and seemed cross when he suspected he was wrong. He was inattentive toward the finish and worried because he wanted to get away by 4.30. The psychologist thought it was a case of lazy mind, made worse by bad suggestions from the parents and too many criticisms.

Psychiatric examination showed the patient to be rebellious, belligerent, blaming others for all his misdeeds, and constantly excusing himself. He liked to make "smarty" remarks, and delighted in repeating to the doctor things he had said to his teacher. On the first visit the doctor advised the mother to get the child to realize that she did trust him, and that because the father did not like him she should let up on criticism and be a little more kind. When he next came to the clinic the question

of school was gone into. He seemed to think the other children picked on him. He felt that parental school was only a threat, and that his mother and father would not let him go there. It was explained to him that his parents could not help it if his instructors so desired, and that, after all, the laws were more binding than his parents. After this the boy began to brag that he could get his teacher "canned" if he wished to do so. It was explained to him that this was only foolish talk and that he really could not do it. It was put up to him that his job was to get through school; then he could go to work and earn money. His father's job was that of barber, and he was doing it well; it was put up to him to do his own job well. He was told about his attitude and it was explained to him that he was covering up his own faults by finding fault with everything else. A week later his mother reported that he was much more contented, and that his attitude had greatly improved since his visit to the clinic.

The social worker in the meantime had called at the home and talked with the father. She had explained to him something of the boy's attitude, and the way he felt about his deficiencies. It was suggested that the Y. M. C. A., or boys club, and class work would be good for Vincent. This suggestion was taken up immediately and he was started at the Y. M. C. A. A week later in the clinic he seemed to be quite antagonistic toward his mother, saying she would not let him do what he wanted to; he was particularly angry because he had forgotten a book which he wanted to bring with him, and she would not let him go back home for it. Another reason for his anger was because she would not let him have coffee. It was explained to him that his surliness and antagonism toward his mother were only to show his superiority, and that it only made him look foolish. It was also explained that athletes did not drink coffee and did not smoke cigarettes. It was then pointed out that he should try to prove his superiority by doing things better than others, instead of arguing and slapping his mother in the face with his bullying remarks. After this the patient was not seen by the doctor for three weeks, but the social worker continued her contact and had him on several outings. On one of these he got very surly and

disagreeable because he wanted to go to a movie instead of to a technical high school.

On the next visit to the clinic the mother was disturbed because he had received only 40 on his report card in deportment. This was no improvement over previous months. She said that when she encouraged him, or bragged about him, he seemed to "go wild." The patient's attitude seemed better, however, and he was quite proud of himself because he had stopped grumbling and being cross when he got up. He said this was due to a calendar he received at Christmas which said "Smile," and he had this where he saw it on awakening. He still thought his teacher had it in for him, and was afraid he would not be promoted again. It was put up to him that it was his fault if he was not promoted, and that the teacher had thought for so long that he was a bad boy that it would require some time to convince her that he had changed.

On his next visit to the clinic, two weeks later, he said he was doing better in school and had received only two bad marks in deportment for that month. He was again told that the other boys were putting it over on him, and causing him to get blamed for things he probably did not do. He suddenly asked his mother how he could control his temper. The doctor then talked to him about how this could be accomplished, and told him how one of the famous golf players learned to control his. Vincent apparently liked to cause an argument between his father and mother, as he told with glee of one they had indulged in that morning as to whether cold or hot water was better for bathing. It was again explained to him that he was doing things to cause arguments between his parents, and that he evidently enjoyed this because while they were arguing they at least were leaving him alone. The mother was again advised to stop criticising him, to encourage him in every way possible, and to get his father also to do this. Following this visit things seemed to clear up completely at home; the patient caused no more trouble, he seemed to adjust himself in school, and began to apply himself in his studies. He was promoted at the end of the term, and his mother and father were both delighted with his adjustment.

In the treatment of this case it was necessary to have the boy's father come to the clinic and explain the situation to him. He proved quite co-operative and since then the patient has gradually improved.

These cases have been chosen more or less at random from among those that have gone through the clinic during the past year. Most of them are either straight behavior difficulties, or the physical element is of relative unimportance. Anyway, there are two ways of looking at the relative importance of the physical element: Either we may say that a person can carry his mental and social adjustments more satisfactorily because of his good health, or we may say that the physical side makes very little difference. At least the importance is so indeterminate that we need not count it. Instead of count, "evaluate" would better express the meaning. For example, there are the deformed who seem to be made rather than ruined by their deformities. Weakness is the spur to effort, and the mental side is made strong because the physical is weak. The physical deficiency of itself does not lead to breaks in personality. Perhaps it is merely the way we take the physical trouble that really counts. This may be what is meant by those who say, "It is such a relief to know the truth. I want to know just what is going on, just what to expect." There have been many cases among adults that were considered "nervous breakdowns" that later seemed to have been tuberculosis, in which apparently the physical was the cause of the trouble only because the sick individual did not know what the matter was. Something was slowing them down; they made effort against the burden, could seem to make no progress against their tasks, and so came to think that there was some weakness in their make-up. Had they known that they had an enervating infection, and that there was this legitimate reason for feeling unequal to their tasks, they would have been spared this source of a sense of inadequacy. And since fear, or sense of inadequacy, is the basis of nervousness the break might have been avoided had the struggler only known what made his tasks seem so terrible and his efforts so futile.

Two general trends are to be seen in these cases. Among the

boys the difficulties seem to belong more in the field of competition, and among the girls more in the line of sex. Perhaps this is to be expected, since in later life the activities of men are more in the competitive atmosphere while those of women are essentially sexual—in the sense that marriage, motherhood, house-keeping, and rearing the young are sexual.

These are symptomatic cures where the results are good. The causes of poor adjustment have been sought out and an attempt made to remedy them. Of course, the trouble is usually with the parents, and it has been necessary to try to get them to change their attitude toward the child. However, no matter how faulty the parents may be, the trouble depends on how the child takes them, and so in every case just as much responsibility is put up to the child as is possible. The emphasis is placed on the child.

If there are obvious faults in the parents they are frankly discussed with that parent in the child's presence, and the causative relation of these faults to the behavior disturbance explained as I see it. For example, the matter of the cultivation of modesty producing shame is gone into with good results for both the mother and the child. In some instances this shame element has been especially difficult with the fathers. When the mother has gone home and reported my remarks to her husband there has been an indignant prohibition of further visits to the clinic. Some of these men have been very hard to reach, but as soon as the social worker has been able to get them to come to the clinic and talk with me there has been no further trouble. I suppose it is the fact that another man has talked of sex with their woman that makes much of the trouble, whereas straight talk from man to man is perfectly all right. The same is true where a mother is too strict or too indulgent. For example, in the case of the boy who was misleading his deaf and dumb mother what he was doing and how he was doing it were gone into as fully as possible with the mother and boy together, of course with careful avoidance of anything smacking of criticism.

The avoidance of moralizing is of the utmost importance.

Some persons might object to the amount of time spent in

each of these cases. In some instances it has seemed highly probable that continuance in school and home was dependent upon the change of attitude induced in the child. When a child gets out of either home or school much time has to be devoted to getting it back into its normal orbit. A child with a behavior problem upsets a home or a schoolroom. The mother and father have different ways of handling the child, and so disagree with more or less acrimony; a ferment is loose among the school children. It is easy to show that the time is well spent.

## CLINIC OF DR. JOSEPH K. CALVIN

MICHAEL REESE HOSPITAL

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### ENURESIS IN CHILDREN

ENURESIS, as you know, is a common occurrence in children, most of the cases being nocturnal.

Enuresis is a symptom, not a disease entity. This symptom, however, because it occasions so much discomfort and annoyance to the entire family, may overshadow the underlying or accompanying conditions. However, in order to obtain successful results in the treatment each case requires individual analysis, as the causes may vary considerably in different instances. The same approach that cures one child may aggravate another. Bad methods of treatment may occasionally temporarily succeed, but incidentally may seriously aggravate other symptoms of the underlying condition.

Unfortunately, it is true that many physicians dread to see a case of enuresis come into the office or clinic because of the unsatisfactory response to the usual treatment. Nevertheless, the mother demands that something be done about the distressing condition.

I wish to present to you today the following cases from my Enuresis Clinic, whose common paramount complaint or symptom on first entering the clinic is, or was, *enuresis*, in order to illustrate the various more or less typical clinical types which may be associated with this symptom in children.

First, I will strike an optimistic note by stating that the average physician can, in most instances, easily and successfully carry out, in a few weeks, the active treatment of established enuresis by adhering to certain fundamental principles. Of course the utmost co-operation of the parents is essential. The phys-

ician must have confidence in his ability to cure this condition and *must communicate this confidence to the child.*

In most cases the causes of bed wetting may be grouped under two large heads:

#### GROUP I. DUE TO ORGANIC OR PHYSICAL DISTURBANCES

**Case I. Diabetes Insipidus.**—This patient is a boy aged eight years. He came to my clinic six months ago with the main complaint of nocturnal enuresis for the past two years and frequency of urination during the day. On closer questioning, he admitted being intensely thirsty most of the time and drinking large quantities of water. Upon measuring his urinary output, it was found that he passed about 8 quarts of urine in twenty-four hours. Outside of a low specific gravity 1.003, the urine was negative. His general health was not disturbed and there were no other symptoms or physical or laboratory findings. Restricting fluid intake was unavailing because of the intense thirst. The hypodermic use of  $\frac{m}{v}$  of surgical pituitrin (posterior lobe) twice daily reduced the output to  $1\frac{1}{2}$  quarts daily and the enuresis stopped. He is still receiving these injections, which will be continued indefinitely.

**Case II. Mental Deficiency.**—This boy, aged ten years, entered the clinic with the main symptom of enuresis, diurnal and nocturnal, which has been present since birth. Closer questioning and observation soon revealed that the child was markedly retarded mentally and unable even to remain in school. The enuresis was then merely a symptom of the infantile type of mind and, of course, cannot be cured.

**Case III. Vaginitis and Cystitis.**—Another child, a girl aged seven years, in whom enuresis had been present for the past two years with frequency of urination during the day. Examination revealed a vaginitis (non-specific) and a pyuria, probably an ascending infection from the vaginitis. The vaginitis was treated by 2 per cent. mercurochrome ointment injected into the vagina twice daily and urotropin (gr. v) and ammonium chlorid (gr. x) three times a day by mouth for the cystitis. Within two months both conditions had responded to treatment and the enuresis stopped.

**Case IV. Chronic Interstitial Nephritis.**—This case died a few weeks ago of a chronic interstitial nephritis, a rather uncommon disease in childhood. The first symptom that brought her to the physician was nocturnal enuresis. She was referred to my clinic two years ago because of the development of nocturnal enuresis. She was then eleven years old and, although somewhat undernourished and anemic, presented no other physical findings. The urine contained only a trace of albumin and no casts. Her blood-pressure, however, was 150 mg. and the blood-urea was 60 mg. per 100 c.c. The kidneys were unable to concentrate or dilute properly, and consequently a large quantity of low specific gravity urine was secreted during the night, resulting in the enuresis.

So you see bed wetting may be caused by *organic* or *physical* disturbances, the more prominent among which are (a) diseases of the central nervous system, such as idiocy, cerebral palsy, spina bifida, tumors, etc., (b) internal glandular disturbances, such as diabetes insipidus, (c) urinary changes, as when the urine is too acid, or too large a quantity, etc., (d) diseases of the bladder and kidney, such as infections, calculi, nephritis, etc., (e) local irritations in neighboring organs, such as narrow meatus, phimosis, pin-worms, fissures, vaginitis, etc.

Those many defects in Group I are only infrequently the causes of enuresis (about one-third of the cases) and even when present, merely their correction does not always control the bed wetting because the habit has been too strongly established. Nevertheless, in considering the possible cause of enuresis in any child, the first point to determine is whether or not there is any physical cause for the difficulty. A complete physical and laboratory examination should be made to rule out these physical causes or correct them before placing the case in Group II.

#### GROUP II. DUE TO BAD HABIT FORMATION EITHER FROM LACK OF PROPER TRAINING OR AN OVERSENSITIVE NERVOUS SYSTEM (NEUROTIC OR EMOTIONALLY UNSTABLE CHILD), OR BOTH

**Case I. Nocturnal Enuresis with Normal Urinary Mechanism During the Day.**—This is by far the most common type met with in our clinic. This girl of six years was brought to the clinic three months ago because she wet the bed every night, in fact, several times a night, since infancy. The urinary rhythm during the day was normal—no increased frequency or incontinence. She did not drink excessively of fluids and by measurement had a normal fluid intake. None of her brothers or sisters had enuresis, nor was there a family history of such. (Not infrequently in this type of case there is a familial history of enuresis.) The mother stated and we observed that the child was a nervous, high-tensioned girl who was emotionally rather unstable (laughed and cried easily, had fits of temper, etc.), played very actively and hard to the point of fatigue or exhaustion, and slept restlessly (tossed about in bed, cried out in her sleep, etc.). She was usually happy at outside play, but had an irritable disposition at home, was careful and neat in personal habits, and making good progress in school. No history of masturbation was obtained.

Physical examination revealed a rather thin, undernourished girl, slightly anemic, with a tendency toward vasomotor instability, but no physical or laboratory findings (urine) having any special bearing on the enuresis.

The mother was much perturbed and annoyed by the enuresis, but had lost faith in herself to cure the condition and had given the matter up half-heartedly as "a bad job." She had tried the usual methods of treatment either intentionally or unintentionally, such as *punishment* and threats (arousing fear, actually to the point of its becoming an obsession in child); *shaming* and ostracizing until the child was much depressed when the subject of enuresis was mentioned; showing great *emotional concern* over the mishaps, weeping, coaxing, pleading, etc., with the child (the child then occupied the limelight which all children love); *arguing and rowing* until the toilet became the battleground for discipline; *awakening the child* several times at night which usually establishes the nocturnal urination habit more strongly. Finally, the mother decided that the child had "*weak kidneys*" which she would have to "*outgrow*," this last stand finally convincing the child to take a resigned attitude.

The above represents a fairly typical history of the most common functional or habit type of enuresis. Of course there may be emotional conflicts in her environment, maladaptation to her surroundings, a psychogenic background, peculiar personality traits and various behavior problems involved, which can usually only properly be brought out by a trained child psychologist or psychiatrist. For this reason we refer our clinic cases to the Mental Hygiene Clinic for this sort of investigation. But in private practice the general practitioner cannot refer all his enuresis cases to a child psychologist because of expense or lack of available psychiatrists, etc.

Consequently, in our clinic we have mapped out a system of investigation of outstanding causes, types, and treatment that is usually successful without resorting to any more special procedures than the general practitioner has available in his office.

I have found that the following special Enuresis Questionnaire aids materially in classifying the etiology and treatment and standardizing the cases.

#### ENURESIS CLINIC

##### *Enuresis:*

Nocturnal.

Diurnal.

##### *Age of onset.*

Continuous or intermittent since onset.

Number of nights weekly.

Number of times a night.

How soon after retiring?

Does patient awaken?

Frequency of urination during day.  
 Amount of fluids taken daily and when?  
 Familial (do other children in family have habit?)  
 Heredity—present in parents?

*Previous treatment and methods of control.*

Fluids restricted?		
Awakened at night?	How often?	
Medicines prescribed?		
Punishment?	What type?	
Emotional concern?		
Arguing and rowing?		
Babying?	Shaming?	Rewards?
Attitude of family.		

*Nervous System:*

Emotional condition.		
Nervous high-tensioned type?		
Play.	Active.	Quiet.
Disposition.		
Sleep.	Sound?	Restless?
Sleeping arrangements.		
Masturbation.		
Personal habits—careless and slovenly.		
careful and neat.		
Progress in school.		

*Physical Examination:*

General nutrition.  
 Special factors bearing on enuresis, as phimosis, etc.

*Urine Examination.*

Before discussing the treatment let me sum up the obvious psychic etiologic factors.

1. High-tensioned, emotionally unstable, nervous, restless, very active child—the hyperkinetic type with an oversensitive nervous system. This sensitive nervous system probably tends to respond too quickly to impulses sent to it from the bladder. Overstimulation of this hypersensitive nervous system by over-work in school, extra music, elocution, and dancing lessons, exciting movie and radio programs, too strenuous play, quarrelling in the home, etc., especially when these extra stimulations occur late in the afternoon or evening, certainly contributes as a cause of the enuresis.
2. Fear concerning the ability to control the bladder brought

about by punishment, shaming, and segregating because of wet mishaps which concentrate the child's mind on the difficulties of bladder control. Fear and anxiety of committing the act, the suggestion acting on the child's mind that she has failed to control a nasty situation is a prominent cause of the difficulty and may become an obsession.

3. Antagonism due to the spirit of the training—arguing, rowing, undue and unreasonable discipline and domination, impatience and inconsistency in methods, in short, making the toilet a battleground for discipline. Children then often wet merely to annoy and get their own way.

4. Emotional scenes on the part of the mother or nurse concerning the use of the toilet, such as showing great concern over accidents, weeping, excessive petting and coaxing, etc. Children love emotional scenes which make them the center of attraction, and so wetting may serve to keep them in the limelight, especially the inferiority child who has difficulty attracting attention by the normal means.

5. Excessive "babying" by overaffection, etc., may lead the child to continue the period of infancy and enjoy maternal care.

6. Masturbation may be associated with enuresis, as both conditions are frequently present in the emotionally unstable (nervous) child. Occasionally the suppression of masturbation may cause enuresis to develop as a substitution habit, or suppressing one may cure both. The relationship between the two should be investigated in any case although my experience leads me to believe that the relationship has been much overemphasized.

7. Emphasizing organic ailments to the children or in their presence without sufficient findings, *i. e.*, "weak kidneys" or "weakness of the bladder," etc. The child may feel justified in persisting in the habit because she is sure that she has "weak kidneys" which she will outgrow when a "big girl."

8. Loss of confidence by the child that she can be cured, as usually the child and mother have given up after many attempts at unsuccessful treatment.

The active *treatment* of such an established case as described above can be easily and successfully carried out within a few

weeks' time, but the utmost co-operation of the parents is essential. This "cure" must be carried out by persons who have good control of the child, have confidence in their ability to cure this condition, and can *communicate this confidence to the child*. The parents usually fail to meet these requirements, and so the treatment should be largely in the hands of the physician.

I. Any physical defects should, of course, be corrected or eliminated if possible. As many of these children are under par and anemic, general improvement of the physical condition by proper diet, hygiene, and tonics is indicated.

II. The following printed instructions are given to the mother:

*Directions to Mothers.*—Stop all punishments or any action that will arouse fear in connection with the habit.

Stop shaming.

Stop all arguing and rowing and dominating unreasonably—the question of the use of the toilet should not be a battle-ground for discipline.

Stop all displays of emotional concern and substitute an indifferent attitude. Treat mishaps in a casual and kindly way so as not to concentrate the child's mind on the failures and difficulties.

Stimulate interest in success by much praise, ado, and rewards for dry nights—avoid mention of wet nights. Never express lack of faith in the child.

Keep a gold star calendar of dry nights only.

Stop "babying" the child by overaffection, etc.

Never mention to the child that he has "weak kidneys," etc., or that he will after years outgrow the habit.

*General Rules.*—1. Restrict fluid (milk, water, soup, etc.) after 4 p. m. The evening meal should be light and dry, *i. e.*, cereal or custard or junket, bread, jello, fruit, etc. Avoid coffee, tea, salt, pepper, and condiments at all meals. Especially avoid salt and sweets after 4 p. m., as these increase thirst.

2. Emptying the bladder before retiring and again at 10 or 11 p. m. Be certain that child urinates freely at these times.

3. *Rest:* An afternoon nap if possible; no excitement or high tension after 5 p. m., such as exercise, reciting, competitive games,

loud laughter, movies, exciting radio programs, etc. The child should sit down and play quietly after 5 p. m. The child should not become too fatigued before retiring and should retire early. Elevating the foot of the bed 6 inches is advisable.

III. *Psychic therapy* consists of eliminating the physiologic causes of bed wetting enumerated previously. This is largely accomplished by the "instructions to mothers" just listed. The child must be talked to by the physician something in this wise: That the habit is not so desperately tragic, and be impressed that the trouble always gets well, and that it continues now because he is worried and keeps thinking about it, etc.

After eliminating fear and anxiety and emotional scenes, etc., confidence must be restored in the child that he can be cured, as usually the child and the mother have given up. Build faith that success can be attained and encourage each child. This is accomplished largely by suggestion. Usually some outside source of stimulation and inspiration is necessary. The physician rather than the parent can best build up this faith in the child. The physician must insist to the child that he can cure him, but to drive this thought home in the child's mind it is necessary to perform some striking yet harmless procedure, *i. e.*, a *hypodermic injection of sterile water at the weekly visits to the physician*, with the absolute assurance to the patient that this will result in a "cure."

A visible record of successes is a very good method to help cultivate an atmosphere of optimism and confidence. Have the mother mark the successful days on a calendar with gold stars and give high praise, thus emphasizing the successes with commendation and reward. Leave the other days blank and never mention or indicate these failures. These gold star calendars should be brought to the physician's office with the patient at weekly intervals.

The problem should never be discussed between the physician and the parent in the presence of the patient. In general, avoid centering the enuretic's attention on his problem and shortcomings. Avoid stressing sex in the treatment if possible. Psycho-analysis is rarely indicated except in adolescent children.

IV. *Drug Therapy.*—Most text-books and articles on enuresis have relegated the drug treatment and enuresis to a very insignificant place. Many authors state that the main value of the drug is the suggestion of a cure to the patient and that consequently almost any drug seems to help. I have found in my clinic that drug therapy is of major importance in speeding up the "cure," providing the drug is carefully selected to meet the indication.

One of the fundamental underlying causes of most enuresis is the nervous high-tensioned child. Consequently, I have used sedatives such as phenol-barbital (luminol) in the maximum dose possible without producing lethargy. If the enuresis is purely nocturnal, it is administered at bedtime (gr. i to child of four or five years), if diurnal, it is given in divided doses three times daily. The results have been remarkable. The enuresis has stopped in many of the cases at once, even with the bad material encountered in dispensary practice. The luminol must be continued for at least two weeks until the habit is thoroughly broken, and then the dose gradually reduced and discontinued unless there is a recurrence, when the course of treatment is repeated. This type of therapy tends to disprove the theory that very profound sleep with diminished consciousness of bladder contractions result in enuresis. The phenol-barbital apparently raises the threshold of the nervous system response, so that the anxiety and fear complex and other subconscious psychic factors do not suggest to or intrude upon the deep sleeping brain, and the high-tensioned overresponsive nerves are quieted. This breaking of a vicious circle is especially successful in the nervous child, but is of value in the treatment of all cases. If the first three principles are also carried out the chances of recurrence are slight. (Bromids may be used instead of phenol-barbital or a combination of the two.)

V. Finally, the easiest method of curing this type of enuresis, but seldom agreeable to the mother, is to change the whole environment by removing the child to another home (boarding or nursery school) or hospital (especially in the ward). These bed wetters almost invariably control the habit soon after being

placed there, but must remain in the new surroundings for at least three weeks to effect a permanent cure. Here the motive is that they try to do as the other children do and stand well with their fellows (be approved of). The attendants should, however, take an indifferent attitude toward the bed wetting.

I disagree with certain authorities (*a*) that some normal children over three years of age cannot go through the night and must be awakened or taught to awaken themselves to go to the toilet in order to avoid wetting the bed. This does not cure, but seems to establish the habit more firmly, even though there are not as many wet sheets. A child past three years of age without physical defects should be able to sleep through the night without emptying the bladder after 10 p. m. (*b*) I also do not believe in the child attempting to suggest to himself by repeating "I am not going to wet the bed tonight," etc. This focuses his attention on the matter and arouses his anxiety. (*c*) Some advise making the child responsible for washing the wet sheets and changing his bed. Even though this is not done in the spirit of punishment, but as a natural consequence, it again makes the wetting a paramount matter in the child's life and calls his attention to his failures (a bad policy). (*d*) The possible value of bladder training by interrupted urinating during the day (start-and-stop method), as advised by some authorities, I believe is outweighed by the danger of concentrating the child's mind on his difficulty.

**Case II. Nocturnal Enuresis with Diurnal Frequency of Urination.**—This boy, five years of age, had wet the bed at least once a night since infancy. He also suffered from frequency of urination during the day, urinating at about one-half to one hour intervals (but not wetting his clothes). He did not drink excessively of fluids. There was no family history of enuresis. He was not a nervous type, nor emotionally unstable, played quietly, and slept soundly. He was bright and alert mentally. Physical and laboratory examinations were negative. The usual treatment had been instituted by the mother, ranging from severe punishment early in the condition through many awakenings at night, etc., to a hopeless attitude in more recent times.

I believe that in this type of case part of the difficulty lies in a small bladder capacity due to a spasticity (spasm) of the bladder muscles which facilitates the development of the enuresis habit, although by no means all of these "diurnal frequency" cases have nocturnal enuresis.

The general treatment in this type of case is common with all the functional cases is to instruct the mothers in the psychic "don't's" and institute the general rules as detailed in Case I.

The special drug treatment consists in administering atropin sulphate, gr. 1/300, or tincture of belladonna,  $\frac{1}{2}$  fl. oz., three times daily (large doses for this boy of five years). In a series of observations carried out in our clinic it was found that normal children when taking large therapeutic doses of belladonna urinated much less frequently in twenty-four hours, but passed about the same total twenty-four-hour quantity of urine as when not taking the drug, providing the fluid intake was constant. This is explained by the fact that belladonna (atropin) relaxes the smooth muscles of the bladder and so increases its capacity to hold fluid.

Changing the reaction of the urine, usually from acid to alkaline, by combining sodium bicarbonate with the belladonna is often of distinct value because the acid reaction of the urine may be a factor in continuing bladder spasm. Only if the child is a high-tensioned, nervous type (the above patient was not) should small doses of bromids be combined with the belladonna and luminol given at night.

**Case III. Nocturnal and Diurnal Enuresis.**—This boy is eight years old and is a nervous, high-tensioned, emotionally unstable type, as in Case I. In fact, the history resembles Case I except for the additional feature of diurnal enuresis. In addition to the treatment prescribed for Case I an alkaline belladonna and bromid mixture was given during the day. Inasmuch as this child was so deeply engrossed in his outdoor active play that he failed to respond to nature's warning until too late to reach the toilet, I insisted that the child remain quietly at home until the habit was broken. However, this must be explained to the child as not a form of punishment, but merely a part of the treatment just as the medicine is.

**Case IV. Diurnal Enuresis Without Nocturnal.**—This is a very unusual type and we have seen only one patient with this combination. This boy is eleven years old and until one month ago had wet his clothes once or more every day for the past four years, but never had nocturnal enuresis. He stated that the desire for urination was followed so quickly by the act of urination (*involuntary*) that he was unable to reach a toilet in time. There was also increased frequency of urination during the day, but no pain (*dysuria*). No other members of the immediate family have enuresis. His emotional make-up appeared normal except that he was depressed by his inability to control the urine. He is very alert mentally and exceptionally bright, but not abnormally neurotic or high tensioned; in fact, he is studious and quiet. He is careful and neat about his appearance. The physical and laboratory examinations were negative. The only methods of control until entering this clinic were attempts to discover psychogenic factors or emotional conflicts, etc., by psychiatrists interested in our enuretic problems. The nearer to puberty an enuretic child approaches, the more likelihood of uncovering a real psychogenic complex to account at least for some phases of the enuresis. I cannot in this short paper discuss this phase of the problem. In this particular case

no marked benefit was derived from the purely psychic therapy as the diurnal enuresis continued.

Our enuresis questionnaire disclosed a very significant fact, that the boy was very fond of highly seasoned and especially salty foods, such as herring, etc., and added extra salt to all his food. This capricious appetite made him constantly thirsty, and he drank 3 quarts or more of fluids during the day, probably accounting for the frequency of urination. His inability to refrain from urinating until a toilet could be reached might be accounted for by a vesical spasm resulting from a non-specific urethritis, from which he suffered four years ago just prior to the onset of the enuresis.

The treatment then was largely to eliminate salt from his diet, restrict his daily fluid intake to normal amounts, control and relax the sudden vesical spasm with large doses of atropin, and changing the reaction of the urine to alkaline. The condition responded readily to this treatment and the boy is now cured of his diurnal enuresis.

This is a borderline case between the organic and functional groups.

**Case V. Nocturnal Enuresis in the Indifferent Type of Child.**—This girl is ten years old. Her mother brought her to the clinic three months ago because of nocturnal enuresis at least once a night since infancy. There was never diurnal enuresis nor any abnormal pain or frequency of urination during the day. Her fluid intake was not abnormal. Her sister, aged eight years, was also an enuretic of the nocturnal type. An uncle had been an enuretic until about twenty years of age. The patient was rather a lethargic, lazy, indifferent type, happy disposition, and careless and slovenly in appearance. She made average progress in school. She slept very soundly at night in the same bed with her enuretic sister. Physical and laboratory examinations were negative, the child appearing in good health. The mother had half-heartedly tried various methods of control from punishment to restricting fluids to patent medicines. She admitted, however, that inasmuch as her brother and two children were bed-wetters that "weak kidneys" must run in the family and she must wait patiently until they outgrew it just as her brother had done. This idea was frequently expressed before the children.

The patient presented an unusual attitude for an enuretic. Most of them are downcast, depressed, and discouraged because of failure to control the bladder and express a great desire to stop if possible. Their enthusiasm can easily be aroused. This patient was happy and seemed delighted by the attention she attracted because of bed-wetting. She was absolutely indifferent to any suggestions offered for treatment—in fact, laughed at the idea of trying to stop. Our usual methods of treatment failed completely. The younger sister of eight years belonged to the first type and was anxious to stop. She was cured merely by having her sleep in a separate bed from her sister, although all other methods as outlined in Case I had failed to influence the bed-wetting previously.

The older sister was sent to the general ward of a children's hospital. The bed-wetting stopped immediately, as she was probably aware that the other children in the ward would discover that she was a bed-wetter. Whereas she had enjoyed the anxiety of adults over her enuresis, she avoided the

derision of girls of her own age by stopping. She was kept in the ward for three weeks until the dry habit had been firmly established. Less time than this often results in a recurrence after returning home. Both children are now free from enuresis which had resisted all other treatment. Camps or boarding schools often act in the same manner to control enuresis.

The surgical treatment of enuresis is in most cases not necessary. Passing of sounds, injection of fluids into the bladder to dilate it, etc., may be dangerous from the standpoint of introducing infection, and should never be done unless a definite surgical indication is present. Inasmuch as most cases can be cured without anatomically correcting bladder defects, these may be considered as overrated in their importance numerically.

I believe that I have presented to you today enough different types of cases for you to appreciate that in order to successfully treat enuresis the individual case must be analyzed and treated individually according to the indications, and that the successful treatment is by no means difficult nor beyond the means of the general practitioner. To see these little patients converted from depressed, downcast, ostracized enuretics to happy, smiling children again is enough compensation for the effort.



## CLINIC OF DR. ROBERT W. KEETON

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### THE MANAGEMENT OF A CASE OF HYPERTENSION AND CHRONIC NEPHRITIS

It is the purpose of the clinic today to present a rather detailed study of the management of a case of hypertension and chronic nephritis. For convenience of presentation the management is divided into three periods.

**Period I.**—This period consisted of seventeen days' stay in the hospital (May 10 to 27, 1926), at which time the details of the diagnosis were established, dietetic management instituted, and the patient recovered from a transitory cardiorenal failure.

**Period II.**—Duration fourteen months, from June 1, 1926 to August 1, 1927. From June 1, 1926 until January 1, 1927 he remained on management. From January 1 until August 1, 1927 he gradually escaped from management until he was overtaken with symptoms suggestive of a cerebral accident and renal insufficiency.

**Period III.**—This period consisted of six months (August 1, 1927 to February 1, 1928) during which two distinct types of dietetic management were used. The results are contrasted.

**Period I.**—The patient, a white male, aged forty-seven, first presented himself for examination May 9, 1926 and was hospitalized May 10, 1926.

*Complaint.*—Cough and hoarseness for eighteen days; heart consciousness, two months; shortness of breath, two months; easily fatigued, two months; constipation for several years, and abdominal distention and gas for twenty-one days.

*Past History.*—The important events may be summarized chronologically as follows: Eight years of age, chorea; fourteen years of age, typhoid fever; twenty-four years of age, jaundice; twenty-five to forty years of age, recurrent periodic headaches, diagnosed as migraine. Three attacks of peri-

tonsillar abscess. Since thirty-seven years of age under observation for chronic nephritis. His co-operation was poor. Blood-pressure was known to be elevated for ten to twelve years.

Venereal infection denied.

*Marital History.*—Two healthy children. His wife has had no spontaneous miscarriages.

*Habits and Mode of Life.*—The type of life led by this patient is most interesting. In college he was an athlete, entering into all forms of sport, but he enjoyed most of all boxing. During the summer months and in the years immediately following college, when bicycling was popular, he indulged in this. He did a great deal of endurance racing. Since he has been in business he has continued his athletic activities. His summer sports have included in serial order, baseball, tennis, and golf. In the winter he has taken daily workouts in the gymnasium and would finish off with a snappy game of hand ball. If he did not develop as vigorous a sweat as he thought that he should have he went into the turkish bath or electric cabinet and then finished with a massage. If he felt below par physically, he spent a little more time in the gymnasium and in the electric cabinet and with the masseur. This usually restored his sense of well being. Only at irregular intervals has he consulted a physician when some major physical embarrassment (peritonsillar abscess) presented itself.

He was an intensive worker and spent most of his day fighting with people. As the engineer of a large architectural firm it was his duty to supervise the engineering details of their buildings, and, above all other things, to see that no labor difficulties arose on the job. He takes pride in the fact that such difficulties have never arisen on any of his buildings. He regularly had two stenographers to whom he dictated alternately, and almost always had from four to five men waiting for interviews.

His evenings were frequently spent at work because at this time he could be free from conferences. He was a social drinker and often drank to the point of inebriation, but was always able to go to work the next day as fresh as ever. He enjoyed food, ate heavily, and delighted in his exercise because it gave him a good appetite.

*Examination.*—Weight 212 pounds; height 5 feet, 4 $\frac{1}{2}$  inches; temperature 100° F.

Pupils equal, regular, react to light and accommodation. Frontal sinuses transilluminate well; left maxillary darker than the right. Ear drums normal. Teeth questionable. Tonsils present; pillars injected. Pharynx red.

Chest: Coarse bronchial râles heard everywhere. Heart enlarged 2 cm. to the left. Systolic blow at apex. Second aortic sound accentuated. Rate 60.

Abdomen distended, tympanitic; liver border down three and one-half to four fingers; spleen not palpable.

On rectal examination prostate found to be normal in size and consistency; no palpable hemorrhoids.

There is slight edema of feet and ankles.

All normal reflexes are present and there are no pathologic ones.

Blood-pressure is 210 systolic and 110 diastolic.

*Laboratory Reports.*—A single specimen of urine shows specific gravity 1025, a trace of albumen, and 4 to 6 granular casts to the low-power field.

Blood examination shows 85 per cent. hemoglobin, 4,940,000 red cells, and 10,550 white cells. Differential: Small lymphocytes 29, large lymphocytes 9, and polymorphonuclear 62.

Blood chemistry: Non-protein nitrogen 85 mgm.; uric acid 2.9 mgm., creatinin, 1.4 mgm.

Blood Wassermann negative.

Sputum negative for tubercle bacilli.

*Diagnosis.*—From a review of the above data it is evident that this patient is only one of a rather large group of people, many of whom are applying to the doctor for assistance. If the doctor happens to be an internist he will recognize the patient as one of the commonest types of office patients. This may be accounted for by the fact that the general practitioner is only too glad to refer such patients elsewhere because of the unsatisfactory methods of treatment available. The patient then has a hypertension, a hypertensive heart, and very definite evidences of chronic nephritis with nitrogen retention and circulatory inadequacy. He also has an acute upper respiratory tract infection which is responsible for the present kidney and circulatory incompetency.

*Management.*—How is this case to be treated? I will say that there is no specific treatment for such cases, but there is a definite method of management, and that this case was selected so that emphasis may be placed on such a management.

In the first place, such cases deserve a period of hospitalization, during which they can be studied, their metabolic balance be restored (if this is possible), and during which they can be educated in their diet and in the limitations which this disease has set upon them. Successful management can continue only through the patient's active and intelligent co-operation. With such co-operation they may often lead lives free of symptoms for long periods of time.

*Upper Respiratory Infection.*—On entrance to the hospital the severity of the infection first claimed our attention. Further study showed the presence of a left maxillary sinusitis with a symptomatic bronchitis. This was treated conservatively until

the patient's condition improved sufficiently to allow drainage by the establishment of a small opening beneath the turbinate.

*The Diet.*—What type of diet shall we order for the patient? He has a chronic nephritis and now a nitrogen retention. The natural answer would be to cut the protein in the diet to the lowest level, since the patient is apparently unable to excrete it completely and on time. Because my experience and that of others has shown that hypertension in many obese patients is benefitted by reduction in weight, I decided to ignore the nitrogen retention and to give this man a diet inadequate in calories with a moderate elevation of protein so as to facilitate his weight loss. It was assumed that if a state of partial under-nutrition could be established this state might result in a more complete oxidation of the nitrogen metabolites and thus prevent their accumulation. The diet order as sent to the dietitian was as follows:

Carbohydrates.....	117
Protein.....	82
Fat.....	44

Making a total of 1196 calories.

The patient's basal requirements were 1880.

In the presence of his restlessness and infection it is safe to say that his actual requirements were basal plus 20 per cent. or 2256. Hence, the 1196 calories represented a caloric under-nutrition of 1060 calories, or 46 per cent. It is usually stated that it is not safe to maintain a normal individual for any length of time on less than 0.5 gram of protein per kilo body weight. This would require that he be fed 49 grams of protein. The diet order was for 82 grams, or 0.84 grams per kilo body weight. This quantity, although definitely above the patient's requirement, is not high. The diet order was filled as follows:

<i>Breakfast:</i>	
10 per cent. fruit.....	100
Toast.....	20
Egg.....	1
Butter.....	5
Milk.....	100
Postum.	

*Lunch:*

Cottage cheese . . . . .	100
Bread . . . . .	20
5 per cent. vegetable . . . . .	200
10 per cent. fruit . . . . .	100
Butter . . . . .	5
Buttermilk . . . . .	200

*Dinner:*

Lean meat . . . . .	100
Bread . . . . .	20
5 per cent. vegetable . . . . .	200
10 per cent. fruit . . . . .	100
Butter . . . . .	5
Buttermilk . . . . .	200

*Bedtime Lunch:*

15 per cent. fruit . . . . .	100
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This will serve as an indication of the menu, although the above schedule was varied slightly to meet the suggestions of the patient.

*The Bowel Movements.*—The patient entered the hospital complaining of abdominal distention and constipation and during his early stage he was incessantly demanding a bottle of citrate of magnesia. Certainly we have been taught that in the face of a failing kidney function the bowel should be made into an excretory organ by daily doses of saline cathartics. On only two occasions during his stay in the hospital did he receive a saline cathartic. During the remainder of the time the bowel movements were secured by altering the character of the diet, making it more bland, since the colon was really in a spastic state, and by the oral and rectal use of mineral oil. This adjustment certainly was important in eliminating the so-called migraine headaches from which he was supposed to be suffering.

*Summary of the Patient's Condition on Discharge Seventeen Days after Admission.*—Weight showed a loss of 20 pounds. Left maxillary sinus was draining freely. One devitalized tooth was removed. Gastro-intestinal symptoms absent, regular bowel movements by means of diet adjustment. Kidney function improved sufficiently so that nitrogen retention is absent. Cardiac compensation established without the use of digitalis. Blood-

pressure on entrance, systolic 210; diastolic 110. This fell rapidly during the time he was in bed to systolic readings of 140 to 160. On day of discharge after he had been an ambulatory patient for ten days, systolic reading of 190, diastolic of 105.

*Subjectively.*—The patient stated that he had not felt so well in years.

**Period II.**—This period lasts from the time of his discharge from the hospital May 27, 1926 until August 1, 1927.

He was under rather close observation until about January 1st. During this time he had the remainder of his devitalized teeth removed and his sinuses cleared of infection. Although he was advised to have a tonsillectomy, he elected to retain the tonsils. He adhered to his diet rather successfully, although he did not weigh his food since his weight gradually decreased from 190 to 160 pounds. His systolic blood-pressure during this time varied from 175 to 185 with an occasional reading of 190. On three occasions it was recorded as 145, 160, 165, with corresponding lowering of the diastolic pressure. He returned to his golf and on some days played 36 holes. He said that he never worked so efficiently as he was doing at this time. In his enthusiastic manner he said that he considered himself a "superman." About January 1, 1927 he ceased reporting to the office, began to eat and drink more liberally, and to work harder. About the first of August, while playing golf on a hot day, he felt a sudden sharp pain in his head associated with dizziness and headache. In my absence from the city he sought other medical advice.

**Period III.**—This period lasts from the time of the incident on the golf course until February 1, 1928. The diagnosis made by his medical attendant was a cerebral hemorrhage without localizing signs. The blood showed a non-protein nitrogen of 56; the urine a trace of albumin and a few hyaline and granular casts. The management of the case adopted is that frequently followed, but one which is very different from that outlined above. His diet was cut to approximately 1000 calories, protein was omitted as far as possible, and salt withheld. He received frequent catharsis, hot packs, and nitroglycerin. After a preliminary period of three weeks in bed he was sent to a sanitarium, where the same management was continued. Here his diet was increased, but the protein and salts were still restricted. After another three to four weeks in the sanitarium he returned to the city and again consulted me. His systolic blood-pressure was 208 and diastolic 112. His weight was 180 pounds. He felt that he had made little or no progress during these six weeks. So on September 14, 1927 I returned the patient to the hospital, changed his diet to carbohydrate 187, protein 67, and fat 64, calories. This diet included meat, eggs, milk, bread, vegetables, and fruits. He was placed in bed and his blood-pressure dropped from a systolic of 208 to 160 with a diastolic of 95 in nine days. There was no retention of nitrogen. It was then decided to remove his tonsils which had been responsible for three peritonsillar abscesses. Following their removal he developed an exacerbation of his nephritis with decreased urinary output and nitrogen retention

in the blood. From this he recovered very slowly. However, he was able to return to work on November 1st, and has been working more and more since that time. His weight has again gradually been reduced to 160 pounds. His systolic blood-pressure ranges from 175 to 185. His urine contains a trace of albumin and a few casts. His Mosenthal test shows a variation in specific gravity between 1008 and 1024. His phenolphthalein test shows an excretion of 25 per cent. the first hour and 15 per cent. the second.

He is living in the hospital, eats two weighed meals a day, and a lunch which is estimated by the eye. He has again returned to an occasional short sweat at the club, walks a stated distance each morning, and dances occasionally in the evening. Mentally he is as keen as ever, but he complains of lack of physical reserve in the evenings. He no longer desires to spend two to three evenings a week playing. However, to the unbiased observer he is making definite progress.

**Principles of the Management.—*Foci of Infection.***—The first objective in the case was to clear up all the foci of infection. These foci were found in three devitalized teeth, in the left maxillary sinus, and the tonsils. It was considered that the eradication of these infections would tend to prevent an exacerbation of his nephritis, and that this eradication would prevent the absorption of materials which must eventually hasten the cardiac failure through their toxic effects on an already overworked myocardium.

**Diet.**—In the second place the diet ordered was always an inadequate one in calories so long as the patient was overweight, and a maintenance one, when his weight had reached its proper level. It was not considered important that this patient should lose any fixed number of pounds or reach any particular weight in a given time. It was considered most important that there be no excess metabolites of any kind in the circulation which the body might store or retain. There must be no smoke in our body furnace. The flame must be so hot that the chimney will be smokeless. For this reason the quantity of protein in the diet was somewhat larger than was actually required, since protein has a definite stimulating effect on the metabolism. No attempt was made to restrict the salts or to see that the diet furnished an alkaline ash.

**Limitation of Activities.**—The third principle of management that is being stressed now is moderation in work and play. He

is to quit before he gets tired. The prolongation of his period of hospitalization has been necessary to insure the maintenance of his diet and the moderation of his work and play.

This principle of diet adjustment has been tried on a number of thin patients with increased blood-pressure. It would seem to be a rational procedure in these cases also, but I have never seen any significant effects on the blood-pressure from its use. Its value, therefore, appears to be confined to hypertensive individuals who are at the same time overweight.

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### THE CLINICAL APPLICATION OF VENOUS PRESSURE DETERMINATIONS

THE study of venous blood-pressure has received attention from a comparatively small group of workers in this country. Hooker and Eyster<sup>1</sup> who have pioneered this field, devised an instrument modeled after that of von Recklinghausen,<sup>2</sup> having for its principle the measurement in centimeters of water of the amount of pressure necessary to cause the collapse of a vein as observed through a glass-covered, air-tight box sealed to the skin over a vein by means of glycerin. With this instrument they established values and variations obtained in the normal individuals. Hooker<sup>3</sup> showed that there was a gradual increase during the day in both ambulatory and bed patients, and further there was an increase after exercise,<sup>4</sup> while physical agents<sup>5</sup> such as heat and cold applied to the hands, while causing a change in the amount of blood in the peripheral veins, caused no corresponding change in venous pressure. Clark<sup>6</sup> as a result of frequent observations in the cases of cardiac decompensation concluded that such determinations were of definite clinical value, in indicating the progress of the condition and the response to therapy, as well as predicting the onset of untoward occurrences. He concluded that readings of 20 cm. indicated a grave cardiac decompensation.

Eyster and Middleton<sup>7</sup> found that the venous pressure never exceeded 11 cm. of water with the patient at rest in bed. They considered the venous pressure to be a measure of the initial load on the right heart, and when this initial load exceeded the ability of the cardiac muscle to respond to this additional

demand on it, by increased efficient activity, decompensation occurs. Elevation of venous pressure precedes changes in the fluid equilibrium. Considering 11 cm. as the critical level between compensation and decompensation, the venous pressure may be used as an index of the degree of decompensation. Readings of 20 cm. and over are found in cases of severe cardiac decompensation.

Repeated estimations, then, in a case of decompensation, measure not only the initial load on the heart, but also determine the ability of the cardiac muscle to respond under stress. This, in other words, is a measure of the reserve power of the heart. A high venous pressure, which remains stationary with the patient under conditions most favorable for the return of normal functional activity of the myocardium, is, therefore, indicative of a poor therapeutic response. Any change in the functional behavior of the myocardium will be immediately reflected in the venous pressure, thus furnishing valuable aid as to prognosis and indications for further therapy. Elevation of venous pressure, while indicating the presence of cardiac failure, does not establish the cause of the failure.

In addition, a local increase in venous pressure occurs as the result of the obstruction to the return flow of blood by pressure on the peripheral vein from tumors, scars, etc. In such cases there is a morbid difference in pressure in both hands.

The following cases exemplify elevation of venous pressure as the result of these two conditions:

The first case illustrates increased venous pressure as the result of the obstruction to the venous return-flow because of cardiac failure. He presents the classical picture of cardiac decompensation. He is forty-seven years of age and was admitted to the hospital ten days ago complaining of marked dyspnea and orthopnea, cough, precordial pain, edema of ankles and legs, which have been present for about eighteen months, but have become so bad in the past two weeks as to force him to seek hospital care. On examination, patient is desperately sick, very dyspneic, each breath is an effort; he is extremely anxious. The eyelids are somewhat edematous, his sclerae subicteric, his lips and the mucosa of the mouth slightly cyanotic. He has marked pulsations in the neck, of the "hopping carotids" type. His chest is barrel-shaped. Expansion is limited but equal on both sides. Tactile fremitus is diminished over both bases posteriorly. Breath-sounds are harsh through-

out and there are a number of fine, moist râles present at both bases. The precordium throbs with each forceful cardiac impulse, the apex is visible and palpable in an area 1 inch in diameter in the sixth interspace—11 cm. from the midsternal line. The left border is 11 cm. from the midsternal line. The right border is beneath the sternum. There is no aortic widening demonstrable. The tones are rapid and loud, with definite systolic murmur at the apex and soft diastolic murmur along the right border of the sternum. The second sound at the aortic is slightly accentuated. The pulses are equal in both arms and are of the water hammer-type. There is a capillary pulse in finger-tips. The liver is 4 cm. below costal margin and is tender, no other viscera or masses palpable. There is edema of the scrotum and pitting on pressure of the skin over the lower part of the back, and along the tibial crests. There are no changes in the reflexes. His temperature is subnormal ranging between 96° and 98° F. His pulse on admission was 120, but under the influence of bed rest and digitalis it has gradually decreased to its present 80. Hemoglobin, 90 per cent.; red blood-cells, 4,750,000; white count, 5300. The urine is dark amber in color, specific gravity of 1.024, contains blood, a 3-mm. ring of albumin, many hyaline and granular casts. Wassermann is four plus, the non-protein nitrogen 50 mg. per 100 c.c. of blood. Icterus index 12, van den Bergh delayed direct, serum bilirubin measures 1.5 mg. per cent. Using bromsulphalein, at the end of thirty minutes 20 per cent. of the dye is retained in the serum. His arterial blood-pressure is 175/70, pulse-pressure 105. The x-ray of his chest shows marked enlargement of heart to both right and left. His eye-grounds show slight edema of the retina and pulsating arteries. His blood-cultures are sterile after seventy-two hours. On admission his venous pressure was 32 cm. of water with heart-rate of 118.

This case, then, is an obvious one of cardiac decompensation. The time of life, the physical findings, the Wassermann, would all indicate a luetic type of heart with an aortic leak as the leading lesion. This high venous pressure determines that the degree of decompensation is very marked. Under the influence of rest in bed and digitalis he seemed to show some clinical improvement during the first four to five days of his stay, his heart-rate diminished to 90 on the fourth day and his venous pressure to 28 cm. of water. Suddenly on the fifth day of his stay in the hospital he experienced a severe agonizing pain in the right chest, and following this his sputum, which had previously been free from blood, became definitely bloody. This of course would suggest a pulmonary infarct, the embolism arising in the right heart and probably having its origin in the usual place—the right auricular appendage—as the result of some fresh thrombotic

process. Subsequently, his venous pressure again increased so that today, his eighth day in the hospital, it is 34 cm. and yet the rate at the apex is 80. This gradually increased venous pressure, with the patient at rest, the most favorable condition for the heart-muscle to respond with increased efficiency, indicates that the muscles is so damaged that such a favorable response is impossible and hence in this case the venous pressure indicates a very unfavorable prognosis.

The next case is that of a white male, thirty-six years of age, a chauffeur by occupation, who was first admitted to the hospital about six months ago. In brief, his history was this:

Five months before admission, while at work, he suddenly had a severe pain in the neck, forcing him to quit work. The following morning his neck was swollen and this swelling progressed for about ten days when it had reached such a size that there was no line of demarcation between face and neck. At the same time he developed a feeling of pressure in the chest which was accompanied by an annoying unproductive cough, aggravated to such a degree by reclining that he was forced to sit up to sleep. He became hoarse and developed some aphonia on exertion. He was gradually becoming weaker and had lost about 50 pounds in five months.

His past history was insignificant, except that he had had a thyroidectomy some 17 years previously.

On examination, the appearance of the patient is striking. His face is cyanotic and "puffy." There is definite swelling of the face, more so on the right, with obvious swelling of the neck. The left palpebral fissure is slightly wider than the right, and there is definite proptosis. Pupils are round and equal and react promptly to light and accommodation.

There is a marked swelling of neck, giving the front part a peculiar flattened appearance. This swelling on the right is hard, slightly movable, and extends from 1 inch below angle of the jaw laterally beyond the border of right sternocleidomastoid muscle. The superficial veins of the chest are markedly dilated and present the classical picture of established collateral circulation between the internal mammary and epigastric veins. Similarly, the veins of the right arm are more prominent than on the left.

Anteriorly, a definite area of dulness can be made out, extending 8 cm. to right of midsternal line and 6 cm. to left of midsternal line in the second interspace. Posteriorly, there is some diminished resonance at both bases with absent fremitus. The heart is essentially negative. The radial pulses are equal. The liver is palpable and enlarged 3 cm. below costal margin. The spleen is not palpable. There is no unusual adenopathy anywhere. Blood-count: Hemoglobin, 75 per cent.; red cells, 3,600,000; white cells, 3750. Differential: Small lymphocytes, 10 per cent.; large lymphocytes, 6 per cent. Polymorphonuclears, 80 per cent.; mononuclears, 4 per cent.; platelets, 300,000. Urine showed no significant findings. The blood Wassermann was negative.  $\alpha$ -Ray of the chest as reported by Dr. Hartung indicated a marked widening of the upper mediastinal space extending especially toward the right. A definite mass could be outlined which showed no pulsation. The venous pressure with the patient at rest in bed was 28 cm. on the right hand and 6 cm. on the left. The biopsy report of a section removed from the mass in the neck indicated a secondary carcinoma. The patient was then put under  $\alpha$ -ray treatment and reported back at frequent intervals for observation. He was readmitted to the hospital about two weeks ago, with all of the previous complaints and findings exaggerated. At this time, however, the venous pressure was equal in both arms, averaging 31 cm. From the physical findings and laboratory results in this case, it is obvious that this is a mediastinal tumor, carcinomatous in nature. On his first admission the venous pressure indicated that the tumor mass was causing pressure on only one of the innominate veins. The development of increased pressure in both hands, then, would indicate that the tumor mass has extended to involve either the other innominate vein or has invaded the auricle itself.

The condition of the patient is very poor. He is unable to lie down. He is most comfortable while sitting bent forward. He is very dyspneic. Each breath causes considerable effort. He has extreme pain in his chest which prevents him from sleeping, and is only partially relieved by opiates. He has an

irritating cough, paroxysmal in character, which causes him severe distress. He is gradually growing weaker. He has no inclination for food, and for the last day or so has had a tendency to become momentarily delirious. The expected outcome will, naturally, occur within a short time.

Subsequent Course: The patient with the cardiac decompensation gradually lost ground clinically and died four days later. At autopsy there was a syphilitic aortitis with eccentric hypertrophy of the heart, especially of left ventricle. Fresh thrombosis of right auricle. Fresh hemorrhagic infarct of lung. Chronic passive hyperemia of lung, spleen, liver, and kidneys.

The patient with the mediastinal tumor gradually became weaker and died two weeks later. The anatomic diagnosis at autopsy, as given by Dr. Jaffe, was malignant tumor of the thymus, infiltrating the pericardium, the wall of both auricles and of the left ventricle, neoplastic lymphangitis in the subpleural lymph-vessels of both lungs, and multiple subpleural metastases. Extensive metastases in the cervical and abdominal lymph-glands, in the thyroid, in both adrenals, in the pancreas, in both kidneys, in the right lobe of the liver. Bronchopneumonia.

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CLINIC OF DRs. KARL K. KOESSLER, SIEGFRIED  
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THE SUCCESSFUL TREATMENT OF SEVERE PERNICIOUS  
ANEMIA

THE treatment of ordinary cases of pernicious anemia with a diet rich in vitamins, and containing liver, has been so generally and successfully adopted that this discussion will only pertain to the most severe cases, *i. e.*, those with an erythrocyte count of from 500,000 to 1,000,000.

These patients complain of anorexia, sore mouth, nausea, vomiting, abdominal distress, and extreme weakness. Most of them also have some edema of the dependent portions of the body; ascites may be present together with an exhaustion psychosis.

It is impossible for patients in this condition to retain sufficient quantities of the usual pernicious anemia diet advocated by a number of people, to benefit them in any way. On the regular régime they become rapidly worse and die.

The sore mouth and the tendency to bleed are strikingly similar to the conditions found in scurvy. The abdominal distress may well be attributed to the achlorhydria and loss of antiseptic qualities of the stomach and small intestine, thus permitting an invasion of the stomach and small intestine by bacterial flora normally present in the colon, and also possibly to nervous impulses arising from the degenerated spinal cord. The nervous symptoms resemble, somewhat, those seen in beriberi and in other cases of vitamin B deficiency.

The logical treatment appeared to be the administration of sufficient hydrochloric acid to overcome the gastro-intestinal symptoms, vitamins in abundance to restore the appetite, re-

move the tendency to bleed, repair the body tissues, and something to stimulate rapidly the production of erythrocytes.

We have found sweetened orange juice, wheat embryo extract (containing 20 per cent. alcohol), and a milk-cream mixture, to be ideal liquid sources of vitamins. The sugar and alcohol rapidly remove the starvation acidosis. Liquid liver extract is an excellent stimulant of the hematopoietic system.

The dietary régime is as follows: First hour: Three ounces of orange-juice to which 30 drops of dilute HCl have been added. This is diluted with water and sweetened to mask the taste of the acid. Second hour: Three ounces of milk and cream mixture (2 ounces of milk and 1 ounce of cream). Third hour: Orange-juice mixture as in first hour to which has been added an ounce of wheat embryo extract. Fourth hour: Milk and cream mixture as above to which has been added  $\frac{1}{2}$  ounce of liquid liver extract, equivalent to about  $\frac{1}{4}$  pound of liver. Fifth hour: Repeat the four-hour schedule as given above. This régime in the hands of a tactful nurse will be properly executed in spirit to prevent an undue loss of rest. Blood-transfusions are indicated in moribund patients unable to take the prescribed dietary régime.

Of the 10 severe patients that we have treated in this way, all have lasting remissions. The following 2 case histories are typical:

**Case I.—History.**—M. S., housewife, aged forty, brought to the ward on a stretcher May 5, 1927, complaining of severe and progressive generalized weakness, fatigue, shortness of breath, numbness of arms and legs, and lemon-colored skin of five months' duration. During the past month she had experienced periods of excessive "nervousness," especially "on an empty stomach" before breakfast and after retiring, which were followed by nausea, eructation and vomiting of small amounts of clear fluid. Practically no solid food was retained for a month and even all liquid food was vomited the last several days. Slight swelling of the ankles and occasional aching occipital headaches had been present for the past few days. She had been confined to bed for seven weeks. During the past eight years the diet consisted chiefly of coffee, rolls and bread, with an occasional egg a day, and bi-weekly small portion of fried meat and potatoes. The patient had been well all her life up until the onset of her present illness, except for influenza in 1918, which confined her to bed for four days. Her catamenia was normal, except for amenorrhea of eight months' duration. There was one healthy child, seventeen years old; there had been no miscarriages. Her husband died at the age of thirty-three years from heart disease. Her sister and

TABLE 1

Date.	Percent-age of hemoglobin.	Red blood-cells, millions.	White blood-cells.	Blood-picture.	Remarks.
5/5/27	30 T.	5.60	5,200	Polychromatophilia, basophilia, anisocytosis, poikilocytosis, normoblasts and megaloblasts.	Nothing retained. Condition grave.
5/7/27	.....	.....	.....	.....	450 c.c. whole blood transfusion direct method.
5/9/27	35	1.22	4,800	Anisocytosis, poikilocytosis, polychromatophilia, megaloblasts, nucleated reds.	Marked improvement. Taking liquid nourishment. Koessler diet.
5/10/27	40	1.20	6,200	.....	Progressive improvement.
5/18/27	45	1.30	4,400	Slight poikilocytosis, polychromatophilia, anisocytosis and achro-mia.	.....
5/23/27	55	2.80	.....	.....	.....
5/30/27	60	2.65	5,400	.....	Spleen felt one finger's breadth below costal margin.
6/1/27	70	3.29	5,500	Poikilocytosis, anisocytosis. Turk-irritation forms.	.....
6/9/27	80	3.26	8,500	Few myelocytes. Slight anisocytosis and poikilocytosis.	.....
6/22/27	80	3.21	6,600	.....	Up and about. Tongue not smooth.
6/27/27	78	4.06	14,400	Reticulated cells.	Optic disks normal.
7/4/27	90	4.30	8,200	.....	Discharged 7/2/27.
7/8/27	81	3.65	8,000	0.5 per cent. reticulated reds. Poikilocytosis, anisocytosis.	Clinic visits.
7/16/27	84	4.10	.....	No pathologic forms.	.....
7/27/27	85	4.53	8,400	Slight poikilocytosis and anisocytosis.	Returned to her work.
8/3/27	97	4.58	5,400	Slight poikilocytosis and anisocytosis.	.....
8/17/27	100 F. M.	5.23	7,900	No changes.	.....
9/7/27	94	4.17	6,300	.....	.....
9/28/27	94	3.81	9,600	Anisocytosis, poikilocytosis, 0.5 per cent. reticulated reds.	.....
10/12/27	87 F. M.	4.30	8,600	.....	.....
11/2/27	103	3.65	10,600	Moderate anisocytosis.	.....
11/23/27	100	4.92	6,600	Normal.	.....
12/14/27	78	3.91	8,400	.....	Patient had a "cold."
1/4/28	120	5.00	9,000	Anisocytosis and poikilocytosis.	.....
2/1/28	106	6.00	10,300	.....	.....

brother had been "anemic"; both are living. There was nothing else of importance in the history.

Physical examination revealed an adult white female, well developed, poorly nourished, cachectic, and chronically ill. Her temperature was 99.8° F., the pulse 112, and respirations 20 to the minute. The blood-pressure was 144/78. The essential physical findings were icteric scleræ, pale lips and mucous membranes, with a tongue that was pale and smooth, and a moderate cervical lymphadenopathy. There were occasional fine, inspiratory, crackling râles over the bases and apices. The heart's apex was in the fifth intercostal space, the left border was 9.5 cm. and the right border was 1 cm. from the midsternal line. There was a soft systolic murmur over the aortic area and over the apex. The pulmonic second sound was accentuated. The abdomen was soft and flaccid with absent superficial reflexes. The deep tendon reflexes were equally exaggerated. There was no Babinski sign present but a momentary patellar clonus was elicited. See Table 1 for data on the blood. An Ewald test-meal on seven different occasions revealed no free hydrochloric acid. The blood Wassermann reaction was negative. A catheterized specimen of urine had a specific gravity of 1.018, it was alkaline in reaction and otherwise negative. The blood creatinin was 1.5 mg. per 100 c.c. of blood; the sugar was 95 mg. The feces contained no blood, parasites, or ova. Roentgenography revealed a normal chest, cecum, stomach, and duodenum. The stomach emptied rapidly. The greater curvature extended 6 inches below the level of the iliac crests, signifying a material degree of ptosis.

**Case II.—History.**—J. H. B., dentist, aged sixty-two, walked into the hospital after coming from Florida via railroad, complaining of generalized weakness, difficulty in walking, sore tongue, poor appetite, 25 pounds loss in weight, and a yellow tinge to the skin of six months' duration. The patient had been apparently well up until 1923 when he noticed that he had less endurance than formerly. In 1924 it was discovered he had a severe anemia; he was hospitalized, given intensive therapy with arsenic from which he became poisoned. Following treatment he returned to work in 1925. He has not worked since then. A second relapse in 1926 sent him to Florida where he placed himself on a daily diet of a pound of liver, with excellent recovery. However, he developed a marked distaste for liver and refused to continue eating it; a relapse occurred. During the two months prior to the time he was seen by us he had been suffering from "nervousness" and insomnia, sleeping only three hours of the twenty-four, and spending his nights driving or sitting in his car. He became garrulous. His blood-count dropped below 2,000,000, and he was sent to Chicago. His weight was 113 pounds. The family history, except for the death of his mother from carcinoma and his father from Bright's disease, was essentially negative. His usual diet had consisted of tea and toast for breakfast, a sandwich for lunch, and meat, potatoes, and gravy for supper. Fruits in season, bread, and biscuits were readily eaten in large quantities.

**Examination.**—Physical examination revealed an adult white male, sixty-two years old, appearing to be in a poor state of nutrition and chron-

ically ill. His temperature on admission was 98.6° F., his pulse 116, and respirations 24 to the minute. The essential physical findings were a lemon-colored skin, subicteric tinge of the sclera, soft systolic blow over the apex of the heart, flaccid abdomen, and marked swelling of the legs and feet. Laboratory examination revealed a negative urinalysis, achlorhydria, erythrocytes 1,000,000 per c.mm., 20 per cent. hemoglobin, and 4550 leukocytes. There was marked anisocytosis and poikilocytosis.

*Treatment and Course.*—The patient was put upon a high caloric, high vitamin, liver-containing diet, and rest in bed. He vigorously protested against the diet and refused to remain in bed. May 19th he commenced to vomit, became disoriented, apprehensive, and held ideas of persecution. The red cell-count dropped to 950,000 per c.mm. with 18 per cent. hemoglobin. He stated that he would rather die than eat the solid food. May 20th he was partaking of small amounts of liquid diet, as outlined above, with a diminution in vomiting. On the 24th of May he commenced to eat small amounts (8 teaspoonfuls) of puréed vegetables. On the 26th his red count was 1,360,000 per c.mm., with 23 per cent. hemoglobin. His liquid diet was gradually being increased and retained. The erythrocytes numbered 1,290,000 per c.mm. on the 2d of June with 32 per cent. hemoglobin. His diet at this time consisted of three small meals a day with the same quantity of liquid diet at half the intervals. June 9th, the twenty-first day of his diet, his red count numbered 1,930,000 per c.mm., and 38 per cent. hemoglobin. With continued perseverance and tact he gradually was made to partake of solid food such as he had never done during his married life, according to the statement of his wife. June 13th, the twenty-ninth day of his period in the hospital, he insisted on leaving and returned to Florida. There he continued to take the diet. Two months after returning to Florida his red blood-cell count numbered 4,500,000 per c.mm. with 85 per cent. hemoglobin.

#### DISCUSSION OF CASES

There is always some vomiting, even when the patients are taking the liquid diet, during the first few days. After two to four days of treatment the patients no longer complain of sore

mouth and abdominal distress. At this time the patients are encouraged to partake of some form of solid food. The choice of the food is left largely to his or her desire. Occasionally belligerent patients refuse solid food, but these patients make a good recovery on continued liquid diet. Usually within ten days the diet has been voluntarily converted from a liquid to a solid form. By this time the patients relish their food, which can be increased in amounts. An improvement in the condition of the blood usually occurs after seven to ten days. From this period, the patients show a progressive improvement.

It is an established fact that vitamins are essential for life: Vitamin A for growth and tissue repair; vitamin B for appetite stimulation and proper functioning of the nervous system; vitamin C for stimulation of certain vital cellular processes. Vitamin D is a requisite adjunct for normal calcium metabolism (membrane permeability, osseous system formation, etc.). Vitamin E is necessary to adequate function of the gonads and development of embryonic tissue.

A vitamin deficiency leads, sooner or later, to impaired tissue function. This may express itself in a number of different ways, depending upon the character of the deficiency. A change in the blood-picture has but recently been recognized. We have demonstrated the production of an anemia in rats by vitamin underfeeding. This has also been observed clinically in man.

The feeding of abundant vitamins soon removes the anemia. The amount of vitamin necessary to restore an anemic animal to health is very much greater than that required to maintain a well-nourished, healthy animal. This has been found to be true in anemic persons. There can be little doubt that liver extract, prepared by removing most of vitamin A, when fed to anemic patients is followed by a rapid blood-regeneration. Under favorable conditions this effect appears to be permanent. The excellent effect of liver, or liquid liver-extract feeding (from which there has been no removal of vitamins), is dependent upon the high vitamin content and the Whipple-Cohn blood-formation substance. Liver is known to contain an abundant amount of vitamins A, B, D, and E.

A diet rich in vitamins is essential for the successful treatment of pernicious anemia. A slow, but certain recovery may be obtained in the absence of a liver diet. Liver contains something that leads to a rapid blood-regeneration. A combination of this unknown factor with abundant vitamin diet leads to a permanent remission insofar as we are able to judge at present.

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CLINIC OF DR. N. S. DAVIS, III

ALEXIAN BROTHERS HOSPITAL

**A CASE OF AURICULAR FLUTTER, CONVERTED TO FIBRILLATION AND THEN TO A NORMAL RHYTHM WITH PERSISTENCE OF THE RHYTHM FOLLOWING A PROSTATECTOMY**

J. McN., a white male, aged seventy-seven, was admitted to the Alexian Brothers' Hospital October 19, 1927 to the service of Dr. C. H. Solomon. He had

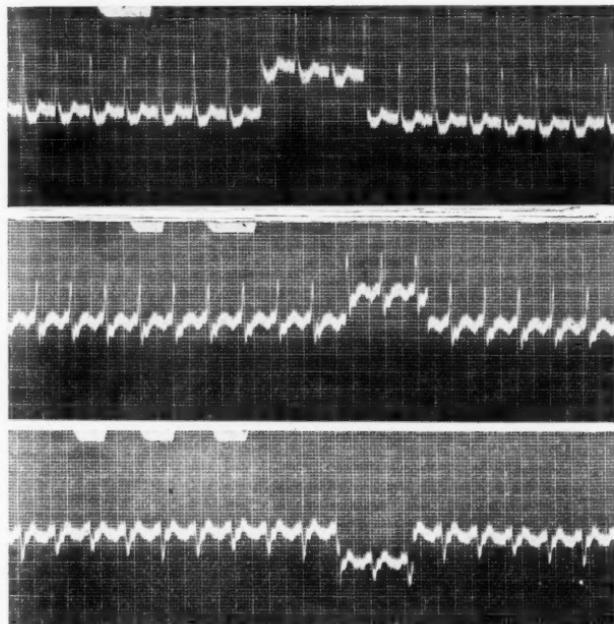


Fig. 33.—Electrocardiogram No. 1, October 24, 1927. Auricular flutter.

had a suprapubic cystotomy in another hospital because of urinary retention due to prostatic hypertrophy. About two years before admission he had first

noted occasional paroxysms of tachycardia which after a period of about six months became persistent, that is, for eighteen months before admission or since the spring of 1926, there had been a persistent regular tachycardia. According to the hospital record, the pulse-rate was 120 on admission. When first seen in consultation, the pulse was found to be regular and 140. The cardiac dulness extended 14 cm. to the left and 4 cm. to the right of the midsternal line; the aortic dulness was 7 cm. in width; blood-pressure 110/80. There were no murmurs. There was some orthopnea, emphysema, a history of marked dyspnea on exertion; roughening of the breath sounds and some coarse bubbling râles; no edema of the extremities. An electrocardiogram

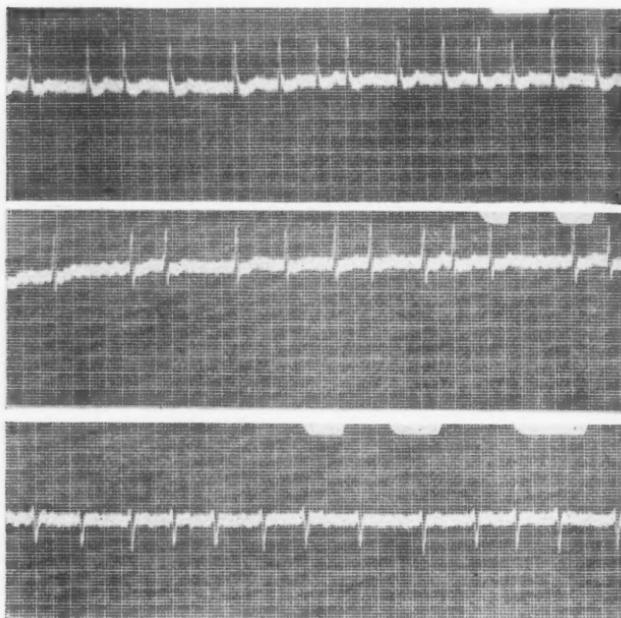


Fig. 34.—Electrocardiogram No. 2, October 26, 1927. Auricular fibrillation.

made October 10, 1927, showed an auricular rate of 300, a ventricular rate of 150; some slurring of the QRS, especially in Leads II and III and an interval of 0.1 second: an auricular flutter with a 2-1 block and considerable myocardial damage.

Prior to the consultation he had been given digitalis, but in such small doses that there was no real effect. Digitalis was commenced in large doses for rapid digitalization (1 cat unit every four hours) on October 24, 1927. On October 25, 1927, the pulse-rate as recorded was 73, but grossly irregular. An electrocardiogram taken on October 26, 1927, was similar to the first

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except that the auricular rate was about 500 and the ventricular from 100 to 120 in the different leads; the flutter had been converted into a fibrillation. Complete digitalization was maintained with 1 cat unit of the powdered leaf daily. October 31, 1927, the pulse became regular. At first the patient objected to another electrocardiogram but after a few days consented and one made on November 3, 1927 showed a normal rhythm; a QRS of not over 0.1 second; a diphasic P in III; slight slurring of the QRS in all leads and left heart preponderance. On November 11, 1927 a blood-chemistry gave sugar

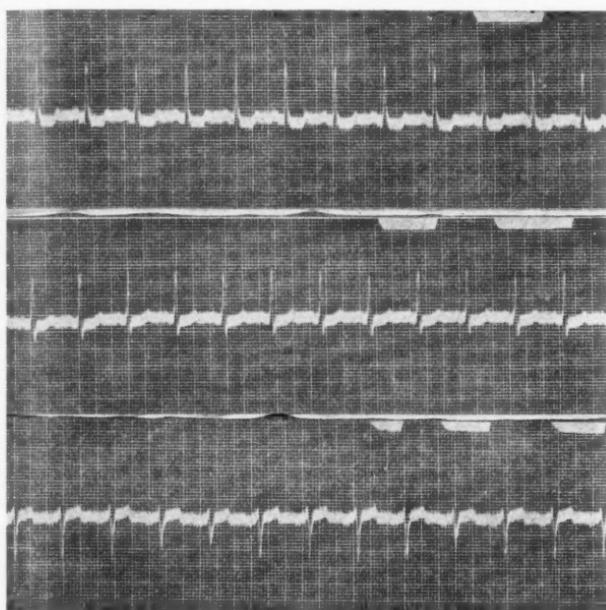


Fig. 35.—Electrocardiogram No. 3, November 3, 1927. Physiologic mechanism, indications of arborization block with considerable myocardial damage. Preoperative.

0.125, non-protein nitrogen 0.039, urea 0.017 and creatinin 0.00195. The pulse remained regular even when digitalis was stopped. On November 12, 1927, a prostatectomy was performed. The pathologist, Dr. J. P. Simonds, reported a chronic prostatitis with marked increase of connective tissue and lymphatic infiltration, moderate cystic dilatation of some of the glands. Following this operation the patient made a rapid and uneventful recovery. An electrocardiogram made December 13, 1927 was essentially similar to the one made November 3, 1927, showing a regular rhythm and physiologic mechanism. On December 21, 1927 was discharged from the hospital still

with the normal rhythm and saying that he was more comfortable than he had been for at least a year and a half, or since the inception of the permanent auricular flutter.

Here we have a case of chronic auricular flutter which on complete digitalization is first converted to a fibrillation and then to a normal rhythm. Before the powdered leaf was administered in large doses, he had been getting 10 drops of a standardized

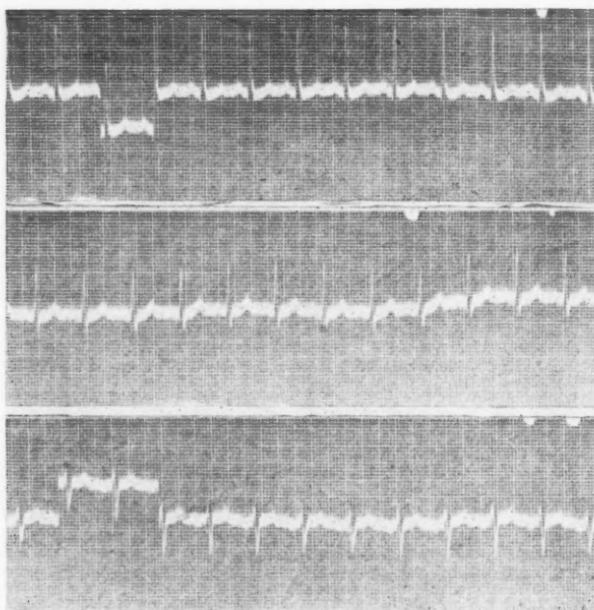


Fig. 36.—Electrocardiogram No. 4, December 12, 1927. Essentially the same as No. 3. Postoperative.

tincture three times daily, and an occasional ampule of digifolin hypodermically, but not enough to really give results. It must be remembered that a drop of a tincture is considerably less than a minim so that not much more than 15 or 20 minims of tincture of digitalis is given when 10 drops of the tincture are administered three times a day. This amount will never produce a real digitalis effect, as is needed in such a condition.

The change from flutter to fibrillation to normal is seen not infrequently and is more apt to occur after digitalization than after quinidin, according to recent statements in the literature. Quinidin therapy was considered in this case, but was not used really because of the long duration of the flutter and the fear that there might be auricular mural thrombi which would break loose and cause emboli in the brain, lungs, and other organs. It was hoped that at least the two-to-one block might be converted to a three-to-one so giving a more efficient ventricular contraction and a greater output. Fortunately in this case, there were no auricular thrombi, so the return to normal rhythm was not accompanied by any distressing complications, but only by a great feeling of relief on the part of the patient.

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## A CASE OF CHRONIC RHEUMATIC HEART DISEASE, MITRAL STENOSIS, AND INSUFFICIENCY WITH AURICULAR FIBRILLATION, CONGESTIVE HEART FAILURE, AND PERICARDITIS WITH EFFUSION, COMPENSATED FOR LIGHT EXERTION AT TIME OF DISCHARGE

J. F., a white man, aged forty-seven, was admitted October 11, 1927 complaining of shortness of breath in the evening of six months' duration; at first, swelling of the feet and ankles only in the evening, but more recently swelling of the legs and thighs as well, which was present all day and night; swelling of the abdomen; difficulty in breathing when lying down. For a few days before admission had been having paroxysms of breathlessness of extreme severity lasting for a few minutes to a few hours. Nocturia was present. These symptoms had developed about six months previously following some heavy lifting, causing at the time a pain in the small of his back.

*Past Illness.*—He had had a severe rheumatism in 1917.

Family history, unimportant. Venereal disease denied.

Examination revealed a white male apparently not acutely ill, but having to sit up in bed, slightly anemic, but not undernourished, slightly cyanotic. Head, neck, mouth negative. Both lungs were resonant throughout, but on auscultation were full of moist bubbling râles with some sonorous râles and prolonged expiratory note. The apex-beat was not visible. There was a systolic thrill at the apex, and the ventricular rate was absolutely irregular. The left cardiac dulness was 5 inches to the left and 1 to the right of the midsternal line. The aortic dulness was within normal limits. There was a late diastolic (pre-systolic), and a systolic murmur. The pulmonary second sound was greater than the aortic. The blood-pressure was 110/80. The ventricular rate was about 120; the radial pulse 64; temperature normal; respiratory rate 28 to 32. The liver border extended three fingerbreadths below the costal margin; there was some ascites. There was a marked edema of the lower extremities, and of the tissues of the back and abdomen and some edema of the scrotum.

*Laboratory Findings.*—Blood (10/11/27): Reds, 4,858,000; whites, 14,600; hemoglobin, 95 per cent.; Wassermann negative. Renal function test, 47 per cent. in three hours. Urine: 1.012-1.026 acid; albumin at two, hyalin casts at one of three examinations.

He was completely digitalized by the large dose method, was given nitroglycerin gm. 00.0006 (gr. 1/100) when necessary

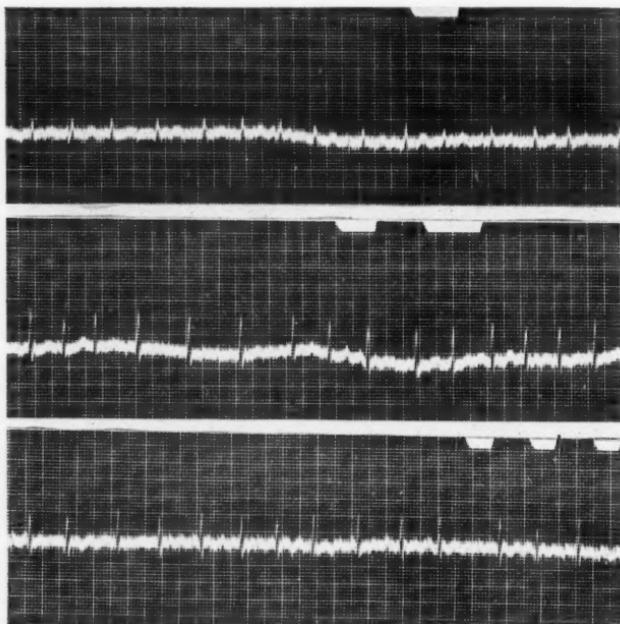


Fig. 37.—Electrocardiogram No. 1, October 11, 1927. Typical auricular fibrillation with a low QRS in Lead I which is also slurred in this lead.

for difficult breathing; Allonal tablets 2, at night, and calcium chloride in doses of 0.6 gm. (gr. x) after meals and was put on a soft diet. The condition did not improve appreciably, though by October 21st he had lost 9 pounds in weight. At this time his urinary excretion varied between 2000 and 3000 c.c. and his intake was in the neighborhood of 1500 c.c. An electrocardio-

gram made the day of admission showed an auricular fibrillation and left heart preponderance. On October 29th, his blood-pressure was 140/80, and he had lost 10 pounds more in weight.

Careful examination on October 24th showed a pericardial effusion which was confirmed by a 2-M. x-ray plate. On October 25th, and daily thereafter for three days, he was given sodium cacodylate 0.45 gm. (gr. viiss) daily subcutaneously. His condition at once changed for the better; the pericardial effusion cleared up; breathing became easier, and the weight loss more rapid. This was accompanied by disappearance of the edema and ascites. By October 28th he was able to be up and around the ward. Improvement continued and on November 5th he was discharged from the hospital weighing 158 pounds, about his normal weight, whereas on admission he had weighed 196 pounds.

While at home he continued to improve and returned to work. Later he stopped the digitalis of his own accord and the symptoms of breathlessness and orthopnea returned and there was some edema of the extremities at night. He developed an acute upper respiratory tract infection and on December 16th was again admitted to the hospital, digitalized and given a cough mixture. The condition cleared up rapidly and on December 24th he left the hospital with instructions to continue the digitalis.

The diagnosis was rheumatic valvular heart disease, mitral stenosis, and insufficiency, congestive heart failure, hydropericardium, auricular fibrillation with compensation for light work when discharged.

The point of most interest about this case was the rapid improvement in the condition when the pericardial effusion cleared up, due to the administration of sodium cacodylate, with no other change in management. Digitalization, which as a rule causes anasarca to disappear in those with congestive heart-failure and auricular fibrillation, and calcium chlorid were of no avail as long as the action of the heart was impaired by the hydropericardium or pericarditis with effusion. Dr. Frank Billings had some patients with pericarditis with effusion when

I was his interne and cleared them up promptly by the administration of sodium cacodylate. He had discovered this action of the drug when administering it for chorea to a patient who had also developed a pericarditis with effusion. I have used it for this purpose many times since, and always with success. A paracentesis of the pericardium will accomplish the same purpose, but is much more difficult than the administration of sodium cacodylate by mouth, subcutaneously, or hypodermically.

Another point to be mentioned is the fact that while there was a history of hard work before the onset of the symptoms, there was at the time of admission a leukocyte count of 14,600 though the temperature was normal. That is, there was distinct evidence of infection present. His relapse occurred without any undue physical or mental exertion, but with an acute respiratory infection. This confirms the ideas of St. Lawrence and others that the onset of heart-failure is almost always associated with infection.

## A CASE OF OBSTRUCTION OF THE RIGHT CORONARY ARTERY, TRANSIENT HEMIPLEGIA WITH MARKED IMPROVEMENT

ON December 25, 1926, W. D. S., a man, aged sixty-seven years, short and stout, had a typical attack of angina pectoris with precordial pain, radiating to the left arm, which was promptly relieved by nitroglycerin. He had no further attacks of precordial distress and continued at work as a business executive, except for a pleasure trip to the West Indies. During the spring of 1927, he developed a slight non-productive cough, but no other symptoms of cardiac failure. In June, 1927, he had another attack of precordial pain with pain in the pit of the stomach and some nausea and vomiting. This pain was but slightly relieved by nitroglycerin, opiates being necessary. With the attack there was considerable cyanosis, some edema of the feet, orthopnea, a pulse of from 90 to 120 which was regular; a blood-pressure of 100/80 (his normal had been 135/80 though there was a history of pressure up to 180 several years earlier); cardiac dulness 13 cm. to the left of the mid-sternal line, 3 cm. to the right, and aortic dulness 7 cm. in width; there were no murmurs or altered heart-tones; the breath-sounds were roughened throughout, but no râles were heard; the urine contained a trace of albumin but was otherwise normal; the temperature was normal. His past history was negative except for a chronic amebic dysentery which had been cleared up by treatment about 1916. A diagnosis of coronary obstruction was made.

Nitroglycerin was prescribed 0.0006 gm. (gr. 1/100) four times daily and theocin 0.3 gm. three times daily or caffeine sodium benzoate 0.45 gm. hypodermically every four hours. Four days after the onset of the coronary obstruction there was a very transient left hemiplegia lasting only a few hours. Two days after this, on the advice of J. B. Herrick, digitalis was commenced by the small dose method, and has been continued ever since (digifolin tablets 1 to 2 daily).

Steady improvement continued though orthopnea was present for about two months. By the end of July, he was able to drive out in the park and on August 4, 1927 an electrocardiograph was made at Alexian Brothers' Hospital which showed an inverted T wave in Leads I and II, and a diphasic T in Lead III; PR 0.2 second; QRS of 0.1 second; notching of RS in Leads II and III; a low QRS never more than 0.8 mvt.; left heart preponderance and physiologic mechanism; arborization block as might be expected with coronary obstruction.

A slow, steady improvement continued until the middle of

December, 1927 when accompanying a gastro-intestinal upset the cough returned and also the orthopnea and frequent premature ventricular systoles were noticed. By this time his pressure had returned to its normal of 135 to 140 and the pulse averaged between 80 and 85. He was able to go to his office for a short time every day and was up and about all day. With the clearing

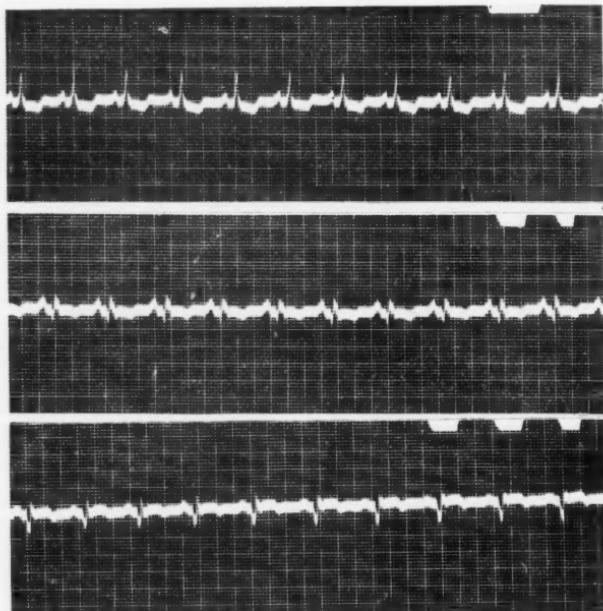


Fig. 38.—Electrocardiogram No. 1, August 4, 1927. Left heart preponderance, arborization block, inverted T wave in all leads. Indicative of serious myocardial damage.

up of the intestinal upset conditions rapidly returned to normal though the premature systoles persisted.

A second electrocardiogram was made at the Alexian Brothers' Hospital December 20, 1927, which showed an inverted T in Lead I except after a premature systole, and in Lead III only following a premature systole; notching of the RS in all leads; QRS duration 0.1 second; left heart preponderance and right ven-

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tricular premature systoles; the QRS deflection was low in II, 0.8 mvt., but normal 1.0 to 1.2 in I and III. Still evidence of arborization block, but a cardiogram that showed marked improvement over the one taken in August. From the type of premature systoles, and from the preponderance as symptoms of pulmonary congestion, cough, and orthopnea, and the absence

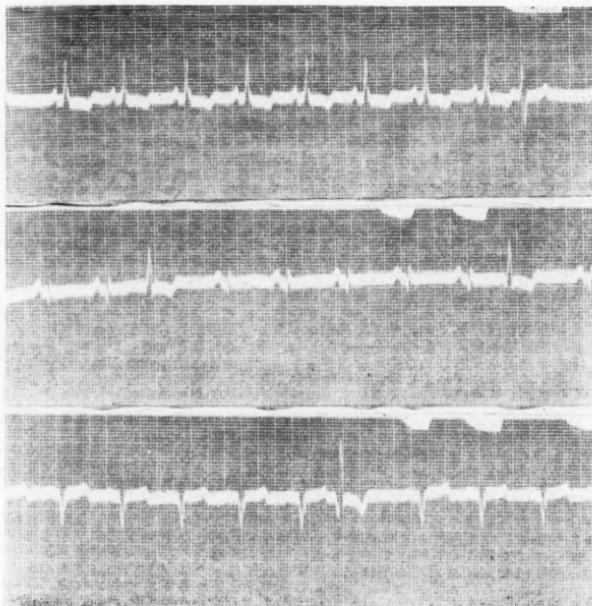
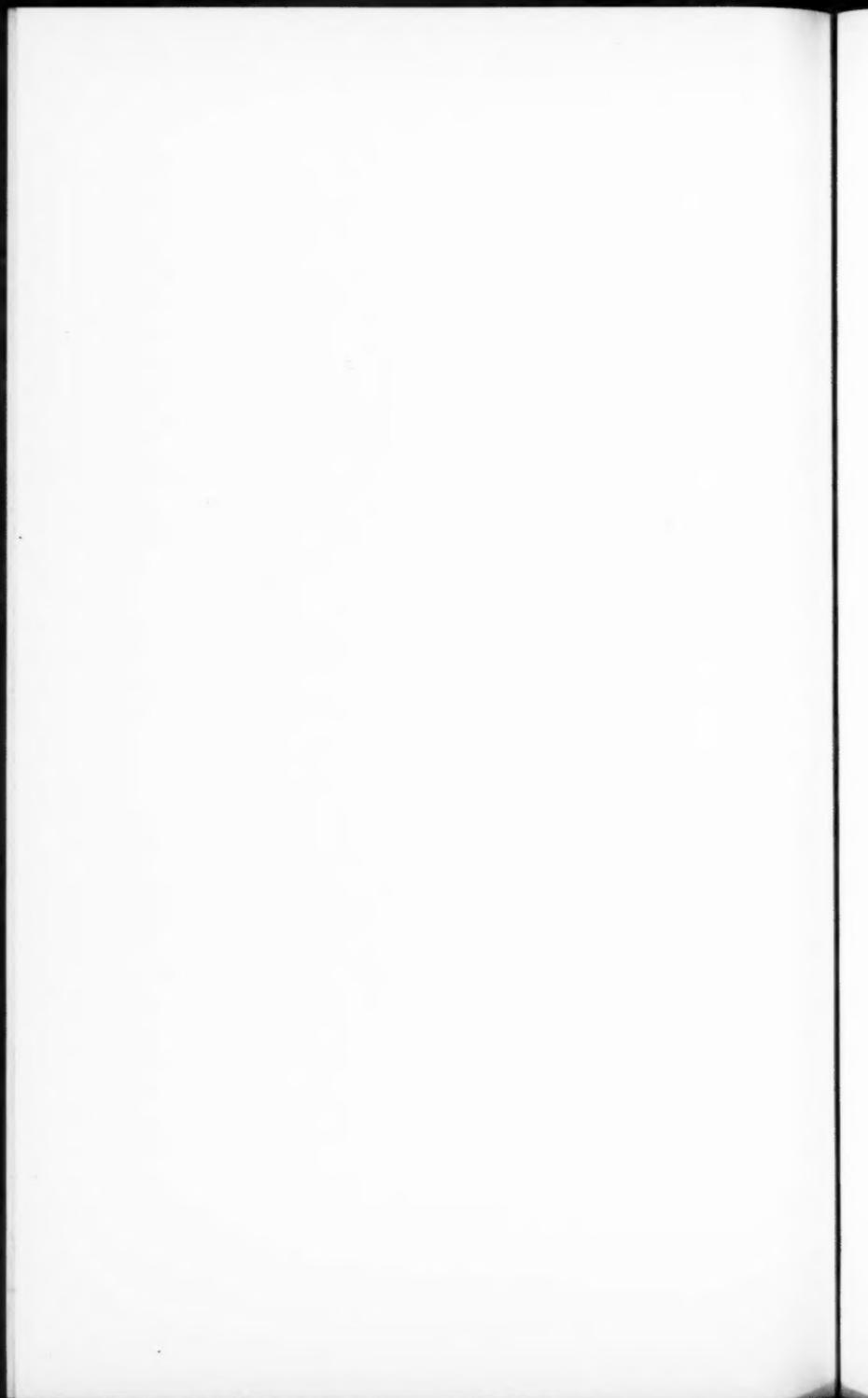


Fig. 39.—Electrocardiogram No. 2, December 20, 1927. Essentially the same as No. 1 except for right ventricular premature systoles and a T wave that is upright except following the premature systoles except in Lead I. Shows considerable improvement.

except at first of evidence of stasis in the peripheral circulation, a diagnosis of obstruction in the branches or in a branch of the right coronary is warranted.

This patient has now gone to San Antonio to escape from the rigorous winter climate and, according to reports, continues to do well.



## A CASE OF ANGINA PECTORIS

In April, 1927, F. M., aged sixty-five, short, heavy set, began to have attacks of precordial pain radiating to the left arm, associated with collapse and fear of impending death. These were brought on by hard work, fatigue, and excitement. The attacks gradually became more severe, came on with less effort, commenced to come on at night without previous exertion or excitement. When first seen on June 20, 1927, was confined to bed because of distress on slightest exertion. There was no orthopnea or edema. The heart dulness extended 14 cm. to the left and 5 cm. to the right of the midsternal line and the aortic dulness was 6 cm. in width. Pulse-rate 84 with occasional premature systoles, ventricular; the blood-pressure was 130/70. The brachial and temporal arteries were tortuous and palpably sclerotic. There were no murmurs or altered cardiac or breath-sounds. Nitroglycerin 0.0006 gm. gave relief if taken every four hours regularly and in addition for pain. In addition to the nitroglycerin, he was given theominal, 1 tablet four times daily. Under this management he continued to improve until he was up and about, walking a good deal without any discomfort. An electrocardiogram made August 10, 1927 showed a physiologic rhythm interrupted by right premature ventricular systoles and an inverted T in II and III only following the premature systoles; QRS 0.1 second and slightly slurred in all leads, especially III: left heart preponderance.

He also had a slight gout, and cincophen caused marked relief. In November theocalcin was substituted for the theominal, and some quinin and quinidin were given because of the frequent premature systoles which at one time caused a pulsus quadrigeminus. On November 20, 1927 he entered the hospital because of a return of the precordial pain following some altercation at home, and remained for two weeks during which period there was no distress. An electrocardiogram made November 21, 1927 showed a physiologic mechanism; rate 100; QRS 0.1 second in II and III, and slightly slurred in all leads; left heart preponderance. Both these cardiograms give slight evidence of arborization block and the right ventricular premature systoles indicate that the right heart is more especially involved in this process. This patient has continued to gain in strength and has been free from discomfort since leaving the hospital. He now takes the nitroglycerin only if there is any distress, but since December 12, 1927 has been taking bismuth subnitrate, 0.3 gm. three times a day in addition to the cincophen and theo-

calcin. According to Stieglitz, bismuth subnitrate is slowly broken up in the intestinal tract with the liberation of nitrites which are steadily absorbed and so should be of value in angina pectoris as well as in hypertension, where Stieglitz has found it to be beneficial.<sup>1</sup>

As to treatment, in both conditions nitrites which cause dilatation of the coronary vessels and so assist in restoring the cor-

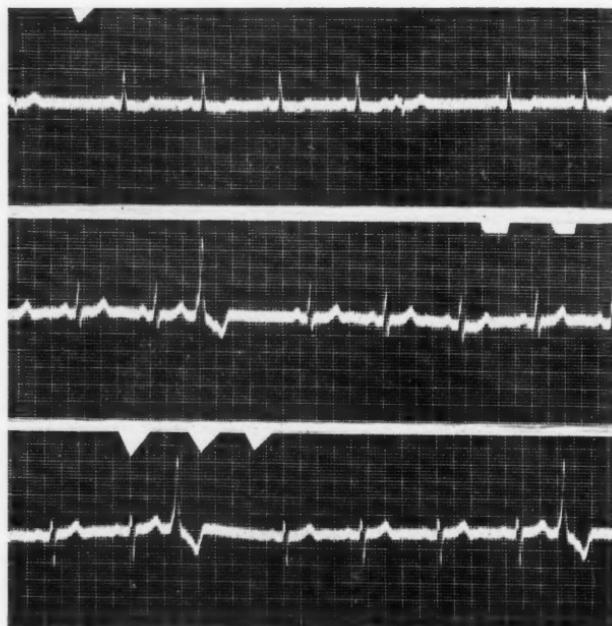


Fig. 40.—Electrocardiogram No. 1, August 10, 1927. Occasional right ventricular premature systoles, left heart preponderance, slight arborization block.

onary circulation are of value. The xanthin diuretics—caffein, theobromin, and theocin—should be and are of great value in these cases as it has been shown that they distinctly increase coronary flow in experimental animals. Of these, from experi-

<sup>1</sup> Stieglitz, E. J.: Bismuth Subnitrate in the Therapy of Hypertension, *Jour. of Pharm. and Exp. Ther.*, xxxii, 1, November, 1927, p. 23.

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mental work, theocin is the most effective, but also the least well borne by the stomach. Of the various xanthin diuretics, theocalcin (theobromin calcium salicylate) is the least irritating and so seems the best to use over a long period. Digitalis may be used in these conditions as it increases the cardiac efficiency. However, it should be given cautiously; while in most kinds of

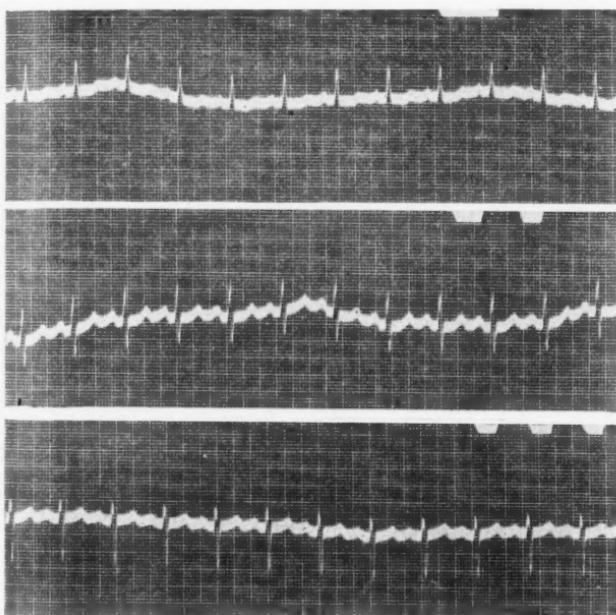


Fig. 41.—Electrocardiogram No. 2, November 11, 1927. Essentially the same as No. 1, though no premature systoles are shown. The higher deflections are indicative of considerable improvement.

heart disease it should be given in large doses that rapid digitalization may be obtained, in these types the small dose method is to be strongly advised. Rest with gradually increasing exertion, mental and physical, is of course an absolute necessity. As to the amount or kind of exercise, that depends entirely on the individual case. Each patient should be allowed a certain amount of latitude, but all must be told that they should avoid

all things that cause discomfort and that they must stop and rest and take nitrites with the first signs of distress.

In both these conditions, the family should be told of the serious nature of the disease and of the possibility of a sudden death at any time, night or day. It is better not to give the patient a full appreciation of the gravity of the situation though the danger of overexertion, mental or physical, should be stressed. Individuals afflicted with either of these diseases should be advised to have all their affairs in shape, to make a will, etc.

These 2 cases, 1 of angina pectoris followed after a period of six months without symptoms by a right coronary obstruction and a transient hemiplegia and 1 of uncomplicated angina pectoris, illustrate the fact that there are exceptions to all rules. Both the above diseases have very high mortality rates and very few cases of coronary obstruction survive for more than a few days. However, at times one may even find one of these in which the area involved is sufficiently small to permit of recovery and healing of the infarct, though of course the functional capacity of the heart is never what it was before the accident. Angina pectoris is not nearly as fatal as generally believed, that is, when the pain is not really due to a coronary obstruction rather than to muscle spasm, and transient ischemia. Coronary obstruction is much more common than is generally believed. The symptoms differ somewhat from those of the true angina pectoris in that, as a rule, there is a reference of the pain down toward the abdomen and an accompanying nausea and vomiting. Probably most of the cases of sudden death ascribed to "acute indigestion" are really due to coronary obstruction. The coronary obstruction may be due to thrombosis or to embolism. A history of hypertension is the rule though the pressure may not have been high at the time of the obstruction. Following the obstruction the pressure is, as a rule, lower than before. Nitrites do not give nearly as much relief in coronary obstruction as in true angina pectoris, though it must be admitted that opiates may be required in this condition also. The recovery after an angina is much more rapid and complete than after an obstruction. In neither case is there necessarily any change in the rhythm.

## CLINIC OF DR. JESSE R. GERSTLEY

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

### BREAST FEEDING

In the last clinic you learned that the modern pediatrician is largely interested in keeping his baby patients well. You learned that he accomplishes this by carefully supervising the diet and by using the various preventive inoculations.

Today I want to review with you in some detail, the question of diet. It is almost superfluous to state that the ideal diet for the newborn baby is breast milk. There is no pediatrician living who will maintain that any modification of cow's milk is equal to that secreted by the human breast. But the modification of cow's milk has been beset with so many difficulties and so many dramatic controversies that medical men have found the subject of sufficient interest to somewhat overshadow breast feeding. For centuries no one has disputed the superiority of maternal nursing, but still, for some reason or other, it has been subjected to very little scientific scrutiny. Due to a rather unusual opportunity I have been able to collect some data which should prove of considerable value.

First, how much food does a breast baby take? A few pediatricians in the past have secured the coöperation of nursing mothers and reported their determinations. However, many observations have been made in the hospital wards and are therefore limited in extent of time. A study of the needs of the infant over a period of months is always welcome. I am glad to be able to present to you the results on two babies of the same mother studied for a considerable length of time.

The mother in question understood the importance of breast feeding. She realized that accurate data were not only interesting but were of value. She had sufficient interest in science to go to the trouble of weighing the babies before and after each

nursing for a period of months. She was of the type met in good class practice, a woman of intelligence, carrying the burden of the usual number of social obligations.

The observations are particularly interesting because the children differed considerably in their make-up. The older child, now three years of age, weighed 7 pounds, 13 ounces at birth. He has a large frame, and is quite chubby. Even from birth he gave the impression of being larger than his weight suggested. Enjoying a good healthy appetite, he gained very rapidly. He has always been in perfect health and is very vigorous, although his subcutaneous tissue and muscles have less tone than the average. He gives the impression of being hydrolabile, although this has been manifested only in one way, *i. e.*, by rapid gains. He has never experienced any severe loss.

The second child, now five months old, weighed 6 pounds, 13 ounces at birth, 1 pound less than his older brother. He has a much smaller frame and gives the impression of being even smaller than his weight. He has much less appetite and gains more slowly. In spite of his slow gain, he never seems hungry, and like his brother is always happy and healthy. His subcutaneous tissue and muscles are of firmer texture.

The tables shown on pages 187 and 188 give the results. Due to lack of space extracts only are given at this time. The record of the first child was not kept in the hospital.

**Discussion.**—Both children were nursed in the hospital, receiving no supplementary feedings. One received seven and the other six feedings daily. Upon discharge from the hospital the mother for her own convenience placed each on five feedings per twenty-four hours.

Scanning these tables, one is impressed with several things. First, it will be noted that in each baby the amounts taken at the individual nursings during the day were quite variable. Text-books often suggest that children take the same amount of food at each nursing. Notice the extreme variability of some of these figures. In keeping with this, note the varying amounts of food taken daily in twenty-four-hour totals. It will be seen that no days are identical.

## BABY P.

Date.	Age.	Weight. gr. lb. oz.	6 A. M.	10 A. M.	2 P. M.	6 P. M.	10 P. M.	Total breast feedings. <sup>1</sup>
1925 Mar. 4	.....	3555						
5	.....	3290						
6	.....	3240						
7	.....	3250						
8	.....	3400						
Apr. 1	4 wk.	9 11						
2	.....	9 11						
3	.....	9 14						
4	1 mo.	9 15	6 L.	?	5	6 $\frac{1}{4}$	4 $\frac{1}{2}$	22 $\frac{1}{4}$
5	.....	10 $\frac{1}{2}$	9 R.	7	(5) <sup>1</sup>	4 $\frac{1}{2}$ R.	7	27 $\frac{1}{2}$ (5)
6	.....	10 1 $\frac{1}{2}$	6 $\frac{1}{2}$ L.	9 $\frac{1}{2}$	4	5	5 $\frac{1}{4}$	30 $\frac{1}{4}$
7	.....	10 1 $\frac{1}{2}$	6 $\frac{1}{2}$ R.	6	5	4	5	26 $\frac{1}{2}$
8	5 wk.	10 1 $\frac{1}{2}$	3 $\frac{1}{2}$ L.	8 $\frac{1}{2}$	5	3 $\frac{1}{4}$	4	24 $\frac{1}{4}$
May 1	.....	11	6 $\frac{1}{2}$ L.	8 $\frac{1}{2}$	3	6 $\frac{1}{4}$	....	24
2	.....	11 1	6 $\frac{1}{2}$ L.	7 $\frac{1}{2}$	4	6 $\frac{1}{2}$	....	25
3	.....	11 2	6 $\frac{1}{2}$ L.	7 $\frac{1}{2}$	(7)	6	....	19 $\frac{1}{2}$ (7)
4	2 mo.	11 3 $\frac{1}{2}$	6 $\frac{1}{2}$ L.	8 $\frac{1}{2}$	3	7	....	25
5	.....	11 2	5 $\frac{1}{2}$ L.	8 $\frac{1}{2}$	3	6	....	23 $\frac{1}{4}$
6	9 wk.	11 1	6 L.	8 $\frac{1}{2}$	4	5	3	26 $\frac{1}{2}$
20	11 wk.	11 12 $\frac{1}{2}$	8 $\frac{1}{2}$ R.	3 $\frac{1}{2}$	5	5	4 $\frac{1}{2}$	27 $\frac{1}{2}$
21	.....	11 13	6 $\frac{1}{2}$ L.	6	5	6 $\frac{1}{4}$	6	29 $\frac{1}{4}$
22	.....	11 14 $\frac{1}{2}$	9 R.	5	6	5	6 $\frac{1}{2}$	31 $\frac{1}{2}$
23	.....	11 15	6 L.	9	4 $\frac{1}{2}$	(7)	9	28 (7)
24	.....	12 $\frac{1}{2}$	7 $\frac{1}{2}$ L.	7 $\frac{1}{2}$	(7)	(7)	6	20 $\frac{1}{4}$ (14)
25	.....	11 14	9 $\frac{1}{2}$ R.	5	?	?	4	17 $\frac{1}{2}$ +
26	.....	12	6 $\frac{1}{2}$ L.	7	5 $\frac{1}{2}$	4	6	29
27	12 wk.	12 $\frac{1}{2}$	6 R.	5	6	4	6 $\frac{1}{2}$	27 $\frac{1}{2}$
28	.....	12 1	5 $\frac{1}{2}$ L.	9	4 $\frac{1}{2}$	?	6 $\frac{1}{2}$	25 $\frac{1}{2}$ +
29	.....	12	6 $\frac{1}{2}$ R.	5 $\frac{1}{2}$	7	6 $\frac{1}{4}$	?	25 $\frac{1}{4}$ +

R. Right breast.

L. Left breast.

Breasts were alternated at each feeding unless otherwise specified.

<sup>1</sup> Figures in this column represent the total number of ounces of breast milk taken. On the days that supplementary feedings were given the number of ounces of bottle feeding are given in parentheses.

## BABY R

Date.	Age.	Weight.	6 A. M.	10 A. M.	2 P. M.	6 P. M.	10 P. M.	Night.	Total breast feedings. <sup>1</sup>
		lb. oz.	6	13					
1927 Oct. 5	.....								
6	.....								
7	.....								
8	.....		2	1½	2	2	2		
9	.....		2	2	2	2	2	2	12
10	.....		2	2	1½	1½	2½	2½	12
11	.....		2	1½	2½	2½	2½	2½	13½
12	1 wk.		2½	3	3	1½	3½	3	16½
Nov. 5	1 mo.	7 15½	6½ L.	4½	4½	3	4½	..	23
6	.....	7 15	4½ R.	4½	(4)†	6 R.	4½	..	18½ (4)
7	.....	8 2	5½ R.	4½	6½	3½	5½	..	25½
8	.....	8 2½	4 L.	5½	4½	4½	4½	..	23
9	5 wk.	8 3½	3½ R.	3½	4½	3½	6½	..	22
10	.....	8 4	4½ L.	4½	3	4	4½	..	20½
11	.....	8 5½	5½ R.	4½	(4)†	5½	5	..	20½ (6)
12	.....	8 6½	6½ R.	3½	3½	3½	4	..	20½
Dec. 5	2 mo.	9 8	5 R.	3½	4½	4	4½	..	21½
6	.....	9 8½	7 L.	4½	(6)†	6	6	..	23½ (6)
7	9 wk.	9 11	6 L.	5	4½	4½	4½	..	25
8	.....	9 8	5½ R.	5	4	(4½)	5	..	19½ (4½)
9	.....	9 8	5½ R.	5½	(6)†	6	5	..	22 (6)
21	11 wk.	10 1½	6	3½ (3)	(6)†	5½ (3)	3½ (3)	..	18½ (12)
22	.....	10 2½	4½ (2)	3 (3)	(6)†	5½ (1)	4½ (2)	..	18 (14)
23	.....	10 7	8	3½ (3)	(6½)†	5 (1½)	3½ (2)	..	19½ (12½)

R. Right breast.

L. Left breast.

Breasts were alternated at each feeding unless otherwise specified.

<sup>1</sup> Figures in this column represent the total number of ounces of breast milk taken. On the days that supplementary feedings were given the number of ounces of bottle feeding are given in parentheses.

One of the most surprising observations is the enormous quantity of milk taken by the first baby at some of his nursings even during the early months of life. I was inclined to insist that some of these were due to errors in weighing, but the observa-

tions were repeated too frequently and with sufficient care to be anything but correct. Once or twice I checked them myself when I happened to be present. As early as the fourth week it will be noticed that this baby took as much as  $8\frac{1}{2}$  ounces and two days later 9 ounces at one twenty-minute nursing. This is entirely beyond any amount which I considered possible for a baby of this age. Notice how much less the younger brother took when of the same age, and again note that the younger brother, even when he reached the weight of the older, still never took such quantities, even though the breasts were full.

These results show what I have felt and repeated so frequently, that it is impossible to standardize the diet of infants. What they take depends primarily upon their own constitutional demands. We should take a lesson from this in bottle feeding. May be it would be wiser to expect the infant to show decided variations in the amounts taken in the individual bottles and in the daily twenty-four hours rather than to expect him to show superhuman accuracy.

In this study the amounts of milk taken from each breast of the mother were also determined. I have indicated when the right breast and when the left breast was used. It is clearly discernible that the right breast was much superior. This was particularly marked in the case of the older child who nursed so vigorously, but it was also true of the younger child. In the latter the figures do not stand out so definitely because the mother many times after using the left breast gave the child a little from the right. We must conclude that although the stimulus of the nursing infant is a great factor in promoting secretion of milk, still there are individual variations in the breasts themselves.

The second baby nursed with so little enthusiasm that he exerted a discouraging influence upon the mother. Though quite conscientious and willing, she did not nurse him as long as the first. Evidently we must still adhere to the time-honored observation that the best stimulus to the secretion of milk is the demand of the nursing infant, though we have just learned that there may be a functional difference in the breasts themselves.

In connection with the subject of breast feeding, relatively

little work has been done upon the chemistry of the breast-milk stool. During the last five years, in connection with Dr. Wang and Miss Wood of the Nelson Morris Institute of the Michael Reese Hospital, I have been making studies along these lines. These studies will be reported in full (*American Journal of Diseases of Children*, in press), but I wish to allude to them just briefly. The object of these studies was as follows:

It is known that diarrheas in breast-fed babies, unless they are of infectious origin, do not disturb the child's health or nutrition. Diarrheas in artificially fed babies are frequently fatal. An occasional study in the past has shown that diarrheas in infants are associated with an increased output of acid in the stools. This acid is generally conceded to be formic and acetic. These acids are produced by the action of the bacteria normally present in the intestine upon the carbohydrate of the food, that is, they are formed from the fermentation of sugar. In the usual mixtures of cow's milk there is about the same amount of sugar as in breast milk. Why is it then that the fermentation in the breast-milk stool is limited and in the cow's-milk stool may go to excess?

In attempting to answer this question we first perfected a technic for the determination of these acids and during these years have made observations upon babies fed with breast milk and with various modifications of cow's milk. We then have added lactose to the breast milk and to the cow's milk to see if there is any difference in the way the sugar ferments in either of these mixtures.

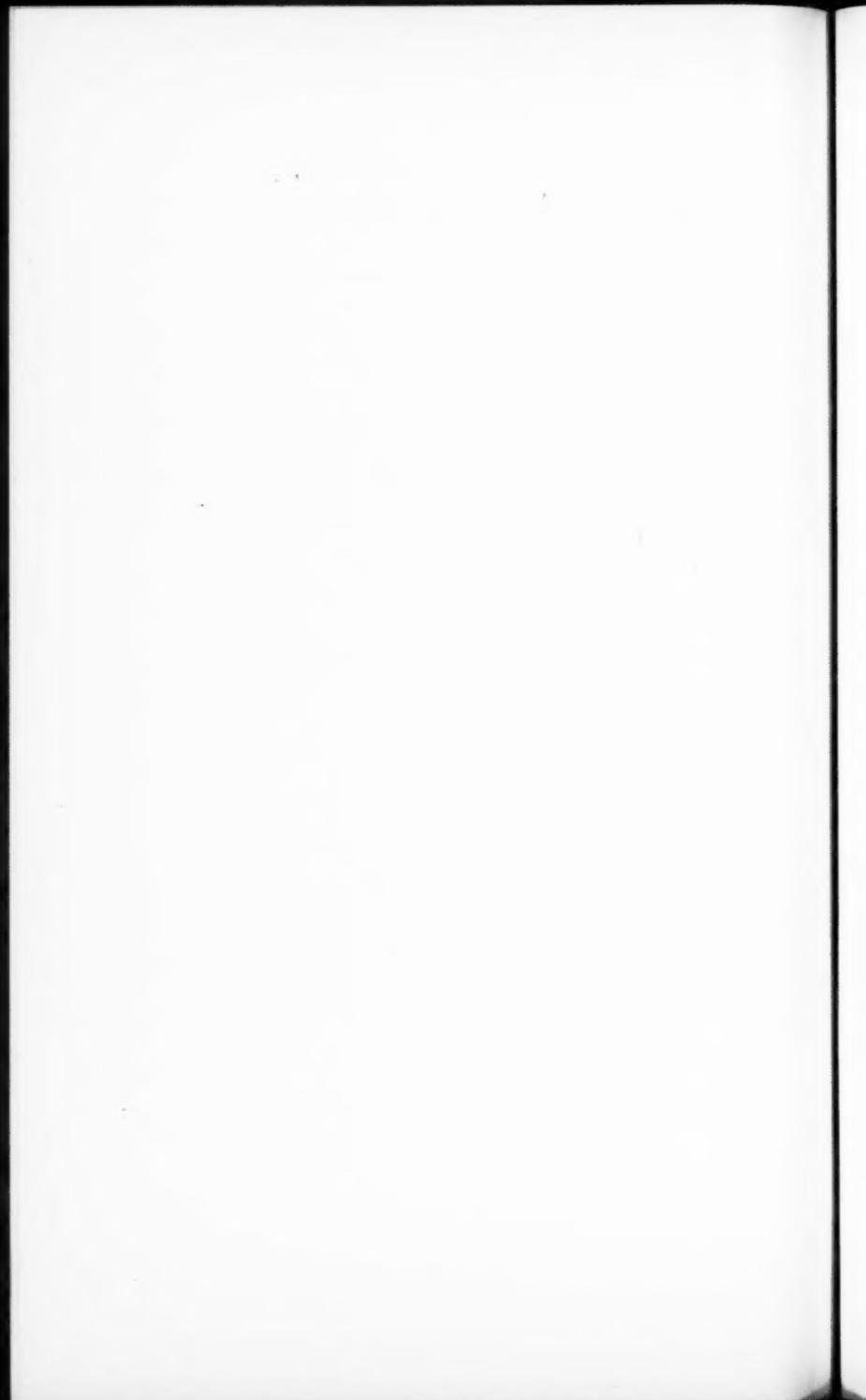
In keeping with the subject of today let me give you some of our results on the breast-fed baby. We found that the acids in the stool of the breast-fed baby are mainly formic, acetic, and lactic acid. Lactic acid is the subject of a study which we have not yet completed, so I shall speak of just the formic and acetic. As a general average, formic acid is in excess of acetic. The amount of formic acid present is represented roughly by 40 to 70 c.c. of tenth normal sodium hydrate. Acetic acid runs from about 20 to 40 c.c. The total amount of combined volatile acids, that is, acids which are either free or held in various combinations in the stool,

is usually below 80 c.c. of tenth normal alkali. There is always a noticeable amount of free acid in stools of the breast fed. This acid, which can be determined by direct titration, is represented roughly by 40 c.c. of tenth normal sodium hydrate. The stools of the breast fed are not very heavy and as we have taken them in metabolism determinations, we find that in a number of experiments the average weight is about 50 gm. per twenty-four hours.

One reads in the text-books about the appearance of the typical "breast" stool, that it is yellow and homogeneous and of pasty consistency. We found in our results that the "breast" stools varied from one in thirty-six hours to four or five daily, that they varied from a hard constipated stool through the condition which is described as normal, down to the green, watery, diarrheal stool containing much mucus and averaging five or more daily. In all these experiments, however, we were impressed with the classical unanimity of the chemical findings. No matter what the appearance of the stool, the twenty-four-hour weight did not show any striking variations and the acid output was relatively constant. In other words, the breast-food stool tends to maintain a certain degree of acidity independent of its physical appearance. This is in striking contrast to the stool of the artificially fed baby, which weighs much more and shows much greater variation in its acidity and chemical composition.

But to return to the breast fed, lactose is supposed to be very loosening. We have performed experiments in which the amount of lactose was doubled and at other times trebled in an effort to see what effect this would have upon the composition of the "breast" stool. To our surprise, the babies showed very little reaction. They remained clinically well, gained rapidly in weight, and the stools were relatively unaffected. This demonstrates the marvelous mechanism existing in the mutual reaction between breast milk and the child's intestinal tract. Everything tends to stability.

The contrast between this situation and that existing in the artificially fed is very striking. I hope to take that up with you in another clinic.



## CLINIC OF DR. HARRY A. SINGER

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### **COLON DISORDERS SIMULATED BY UROLITHIASIS**

ONE of the most frequent presenting complaints encountered in a medical clinic is chronic abdominal pain. The former attempts to account for the distress on the grounds of gastric neuroses or intestinal dyspepsias failed to explain, rationally, the various causes of this common symptom. The more recent resort to appendectomy has demonstrated, judging from the results, that the appendix is rarely the cause of the "chronic abdomen." It had long been known that organic lesions of the colon characterized anatomically by structural changes and clinically by the presence of pus, blood, and mucus in the stools were associated with chronic abdominal distress. However, in most of the cases of abdominal pain no evidence of inflammation as determined by proctoscopic or stool examination was present in the colon. How then was the long-standing discomfort in the cases free from true colitis to be explained? It was not until the profession realized that abdominal distress might be produced not only by organic, but also by functional disturbances of the colon that our nebulous ideas on the subject became somewhat clarified. Sippy repeatedly emphasized the presence of two distinct groups of patients who suffered from chronic abdominal pain, the first, a small group with organic changes (cases of true colitis), and the second a far more numerous group in which the colon was anatomically normal, but functionally deranged.

The soundness of Sippy's teachings coupled with the gratifying response in many cases to "bowel management" soon served to popularize the diagnosis of disordered colon, with the result that "irritable colon" (or spastic or cathartic colitis) has

now become the commonest diagnosis in gastro-intestinal clinics in Chicago today. In fact, the pendulum has swung to the opposite side with such force that in the absence of a clear-cut syndrome pointing to peptic ulcer or gall-bladder disease the presence of a disorder of the colon is immediately invoked to explain the abdominal symptoms. A review of the history charts in practically all clinics leaves one with the impression that the diagnosis of irritable colon constitutes the dumping ground for any type of chronic abdominal pain which is not readily classified under gastroduodenal ulcer or biliary tract disease.

The sufferer of chronic abdominal pain entering a medical clinic is, as a rule, assigned to the gastro-intestinal division where routinely he is subjected to a series of test diets, enemas, and x-ray examinations. In the absence of definite findings the diagnosis of colonic disorder is made. The stools may be hard or soft; the pain may be aggravated or relieved by food-taking; spontaneous evacuation or an enema may ameliorate or augment the distress and still the diagnosis of an irritable colon be compatible. If the patient responds to bowel management it is assumed that the original impression was correct. Failure to respond to treatment, however, is not taken to militate against the diagnosis since not a few bowel cases are notoriously refractory, especially those on a neurasthenic or psychoneurotic basis.

Since the manifestations of an irritable bowel are so protean, and functional derangement of the colon can be produced reflexly, it is no surprise to learn that with the present tendency to look upon indistinctive abdominal distress as colonic in origin a number of extracolonic affections are overlooked. Of the diseases which simulate colon disorders, those of the urinary tract are perhaps the most frequent. I have chosen for today's clinic cases of abdominal pain due to urolithiasis since demonstration of the urinary calculus establishes the diagnosis unequivocally and eliminates the necessity of a lengthy discussion to prove its correctness.

**Case I.**—A. S. H., a medical student, twenty-four years of age, complained of left lower abdominal pain and severe constipation which had been

present intermittently for three months. He stated his trouble started following a picnic at which he participated in the athletic contests of the day and ate freely of "indigestible" foods, including two unripe pears. The pain in the initial attack, which lasted several hours and proved refractory to enemas and catharsis, was greatly ameliorated by heat applied locally. The subsequent attacks occurred on the average of every four to five days, lasting each time from three to eight hours. The pain which was felt deep in the abdomen at a point midway between the left anterior superior spine and the umbilicus always remained strictly localized. It was described as a continuous, dull ache, at times more severe than at others, but at no time intolerable. Heat and rest afforded the greatest degree of relief. Evacuation of the bowel had no appreciable effect upon the pain. No relationship of the distress to food-taking could be inferred. Associated with the attacks of pain was marked constipation. Indeed, costiveness was so pronounced that the patient found it necessary on more than one occasion to remove the stool manually. The feces following an attack were exceedingly dry and hard whereas at other times they were normally soft and moist.

The student sought the advice of one of his instructors who referred him to a gastro-enterologist. Following a thorough examination alkaline powders, which had a laxative effect, and belladonna were prescribed and his diet restricted to bland foods. After following this régime for a few days the pain disappeared. The patient remained comfortable for approximately two weeks, at the end of which time he discontinued the therapy. The abdominal discomfort returned shortly and in spite of resumption of the treatment no relief was obtained. One day the patient suffered a rather severe and persistent attack of left lower quadrant pain which occasioned him to enter the clinic. In addition to eliciting the above information, it was learned that preceding the onset of his last attack of pain he had played hand-ball for the first time that season.

There were no physical findings of note save, perhaps, slight tenderness upon deep pressure over the region of the sigmoid. The possibility of a ureteral calculus suggested itself but inquiry into the urinary history failed to disclose any disturbance referable to micturition. A fresh specimen of urine was obtained and examined immediately. Tests for albumin were found to be negative. In the sediment obtained by centrifuging, a few red blood-corpuscles and a number of leukocytes were found together with many calcium oxalate crystals. A probable diagnosis of ureteral calculus was made and an x-ray of the urinary tract ordered. Flat films of the abdomen failed to disclose any adventitious shadows in the kidneys, the ureters or bladder. The patient was advised to drink large quantities of water, to keep the urine alkaline, and to avoid certain foodstuffs rich in calcium or oxalates. Inasmuch as he was about to commence his internship the patient decided to postpone cystoscopic examination which was suggested at the time.

The young man now returns six months after his initial visit to the clinic with the information that shortly after beginning his internship he had in addition to the mild abdominal pain

which was experienced from the very onset of his illness, a number of very severe colics. The intense pain which never radiated was felt in exactly the same localized area as the chronic discomfort. A second set of films taken two months after the first Roentgen examination disclosed the presence of a shadow in the lower third of the left ureter at a point which corresponded to the location of the pain. A month later a urinary calculus was spontaneously passed since when, the patient states, he has been entirely relieved not only of the abdominal pain but also of the attacks of constipation.

**Case II.**—J. M., a man of fifty-one, sought an opinion regarding the advisability of an appendectomy which was alleged to be responsible for reflex colon pain. He stated that he had been troubled for two years with abdominal distress described as a feeling as though gas were imprisoned and could not pass in one direction or the other. The discomfort was a continuous one and remained strictly localized to a point midway between the costal margin in the left mammary line and the left anterior superior spine. Accompanying the pain was a disagreeable rumbling and gurgling. The distress occurred in attacks, each of which continued, until the lower bowel was evacuated. At the onset the pain appeared at approximately six-week intervals but gradually the attacks increased in frequency until finally the discomfort became an almost daily occurrence. Constipation, for which the patient frequently resorted to cathartics, was marked throughout his illness. Cascara was first employed only to be abandoned later when it began to lose its effectiveness. Citrate of magnesia was then favored and finally resort was had to magnesium sulphate.

The patient stated that as long as his stools were in a liquid or semisolid state he had no abdominal discomfort regardless of the type of food eaten. However, during the period of costiveness the ingestion of certain articles of diet would after one and one-half hours be followed by the typical discomfort. The offending foodstuffs included meat (especially fried), white bread, potatoes, spicy foods, lettuce, various salads, and oranges. If he avoided the above foods, even though constipated he experienced no postprandial pain. The discomfort which occurred persisted until the passage of a stool, usually effected by means of a cathartic or an enema. A clysm led regularly to the expulsion of a fecal ball followed by the passage of gas in large quantities. Not until sufficient time had elapsed for the ac-

cumulation of fecal material did the pain recur. The passage of flatus alone led to only partial amelioration. Unless evacuation was accomplished by catharsis the stool was comprised of small, dry, and hard fecal balls. No beneficial effect was noted from heat applied to the abdomen. Continuous jarring, the result of an automobile ride on a rough road, caused aggravation of the symptoms. No other noteworthy information was obtained except that the patient had experienced peculiar spasmodic contractions in the perineal region some time prior. Following urethrocytoscopic examination he was informed that a slight mucosal erosion was found in the prostatic urethra, which disappeared after topical applications of silver nitrate.

An x-ray report from a reliable laboratory stated that a gastro-intestinal series showed colonic stasis and a long, kinked, retrocecal appendix which was tender. It was explained to the patient that his colon disturbance was reflex from the appendix and on this basis an appendectomy was advised. The surgeon whom the patient subsequently consulted curiously enough declined to operate unless further evidence was adduced to incriminate the appendix. Since the patient failed to obtain relief from various types of bowel management and opinions regarding the efficacy of an appendectomy were conflicting he was at a loss as to what course to pursue. I informed the patient after obtaining his history that extirpation of the vermiform appendix, judging from somewhat similar cases, would in all probability be followed by disappointing results, whereupon he wished to know what other measure I could suggest to relieve him. It was evident that treatment directed toward a functional disorder of the colon offered little hope for alleviation, since several attempts in the hands of competent physicians had failed. If the patient were to be helped it seemed necessary to uncover a cause for the disturbance which had not been investigated previously. Attention was therefore focused upon the urinary apparatus.

A fresh specimen of urine was obtained immediately and submitted to the laboratory for examination with express directions to look for microscopic blood. The presence of red

blood-cells was disclosed and the tentative diagnosis of urethral stone made. The patient was advised to return for x-ray examination of the kidneys and ureters and to submit daily specimens of urine. On the day of his appointment he telephoned that he had been suffering rather severe pain in the same region as his previous attacks and on this account was unable to visit the x-ray laboratory. The following week he appeared in the clinic and presented me with a rough, oval urolith which he stated he passed without any great difficulty five days after his first visit.

Following the passage of the calculus, which occurred a month ago, I advised the patient to stop all medication and to disregard any dietary restrictions. He has discontinued catharsis and enemas, and has been on a general diet without any symptoms whatsoever, either intestinal or urinary, resulting at any time during the past month. Periodic urinalyses have all been negative and a flat plate of the abdomen taken recently shows no adventitious shadows in the urinary tract. The spontaneous passage of the calculus has to all intents and purposes resulted in a cure.

**Case III.**—I. M., a young man, twenty-seven years of age, had been troubled for five years with pain in the right upper abdominal quadrant which appeared only during periods of nervous tension or emotional strain. Unless disturbed psychically no distress appeared regardless of the kind of food eaten or the type of stool passed. The stools which accompanied or followed the abdominal pain were thin, pencil-like, and often fragmented. Otherwise they were of normal form and consistency. No mucus had ever been noticed in the feces. Three and one-half months ago in the absence of any previous mental upset, the patient had cramp-like pain of moderate severity strictly localized to an area the size of a silver quarter, situated just beneath the costal arch in the anterior axillary line, deep within the abdomen. The pain was experienced in the morning upon arising, lasted unabated for an hour or so, gradually became milder, and as noon approached disappeared entirely. During the remainder of the day he suffered from no discomfort. The following morning he arose again with pain which behaved similarly to that of the previous day. The reappearance of the discomfort led him to resort to catharsis. In spite of the absence of constipation and of any nervous strain, the left upper abdominal pain recurred each successive morning for five days, after which time he sought medical aid.

Physical examination was practically negative except for distinct tenderness in the left costovertebral angle upon fist per-

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cussion, and a palpable rope-like sigmoid which was slightly tender. The stool obtained by means of a suppository revealed nothing abnormal. Urinalysis, however, disclosed the presence of many red blood-cells. An x-ray plate taken the following day was reported negative for evidence of urinary calculi. It was decided to await developments before cystoscopic examination was recommended. Following an auto ride four days after the roentgenographic examination the patient developed excruciating pain in the left upper abdomen and passed large quantities of blood per urethram. The following day another colic of equal severity occurred, during the course of which the pain was perceived to pass more and more distally until at the end of eight hours or so no further pain was felt. Unfortunately, no attempt was made to collect the urine or to discover the stone in voided specimens. It is to be assumed that the calculus was passed, for since the occurrence of the severe colics approximately three months ago, the patient has had no left abdominal pain and his urine has been repeatedly negative for microscopic blood. Nevertheless, he continues to have attacks of mild pain in the right upper quadrant and a spastic type of stool whenever he is emotionally disturbed.

**Comment.**—It is a matter of common knowledge among physicians that acute gastro-intestinal disease may be simulated by urinary tract lesions. For instance, it is a widely accepted rule that no patient is to be submitted to operation for appendectomy unless urinalysis has been performed to rule out a pyelitis or other urinary tract affection. However, the fact that urinary tract disease can appear under the guise of the "chronic abdomen" is not as widely recognized by the general profession. Genito-urinary specialists are well aware of this mimicry, and I am constantly twitted by my colleagues practising this branch of medicine and surgery, regarding the ineffectiveness of petrolatum and restricted diets in overcoming various urinary tract lesions which have been overlooked, particularly by gastro-enterologists.

It is not necessary to enumerate the diagnostic features which serve to distinguish the irritable bowel from lesions of the urin-

ary tract. Adequate accounts of these diseases are readily accessible.<sup>1</sup> It is of importance merely to possess the knowledge that chronic abdominal distress may be produced by urinary tract disease and to bear this fact in mind when confronted with a case in which mild abdominal pain is the presenting complaint. The urinalysis should be performed with scrupulous care. Where a urinary calculus is suspected it is important to obtain a fresh specimen and to centrifuge it; otherwise the presence of erythrocytes may be overlooked. It is advisable to test for blood chemically also, since it occasionally happens that the escaped red cells appear in the voided urine in a lake form and the shadowy outlines of the erythrocytes might readily escape detection. Another and perhaps more cogent reason for performing the chemical test is to aid in the differentiation between yeast cells and red blood-corpuscles. Particularly recently has it been necessary to call the attention of our laboratory technicians to the fact that the spherical bodies frequently reported by them as erythrocytes are contaminating yeast cells apparently derived from the glassware or tap water. If the first urinalysis is negative the examination should be repeated a number of times before drawing any conclusions.

The first 2 cases require little elucidation or elaboration. In each case the cessation of symptoms which had been present for months, occurring simultaneously with the passage of the stone, leaves little room for doubt but that a cause-and-effect relationship existed between the presence of the calculus and the abdominal manifestations. It is noteworthy that in both instances obstinate constipation was associated with the attacks of pain indicating a reflex effect upon the overlying colon. Indeed, in the second case the symptoms, with the exception of the colic associated with the actual passage of the concrement, were for two years entirely colonic since the distress occurred only during periods of constipation, and relief was regularly obtained by evacuation of the bowel.

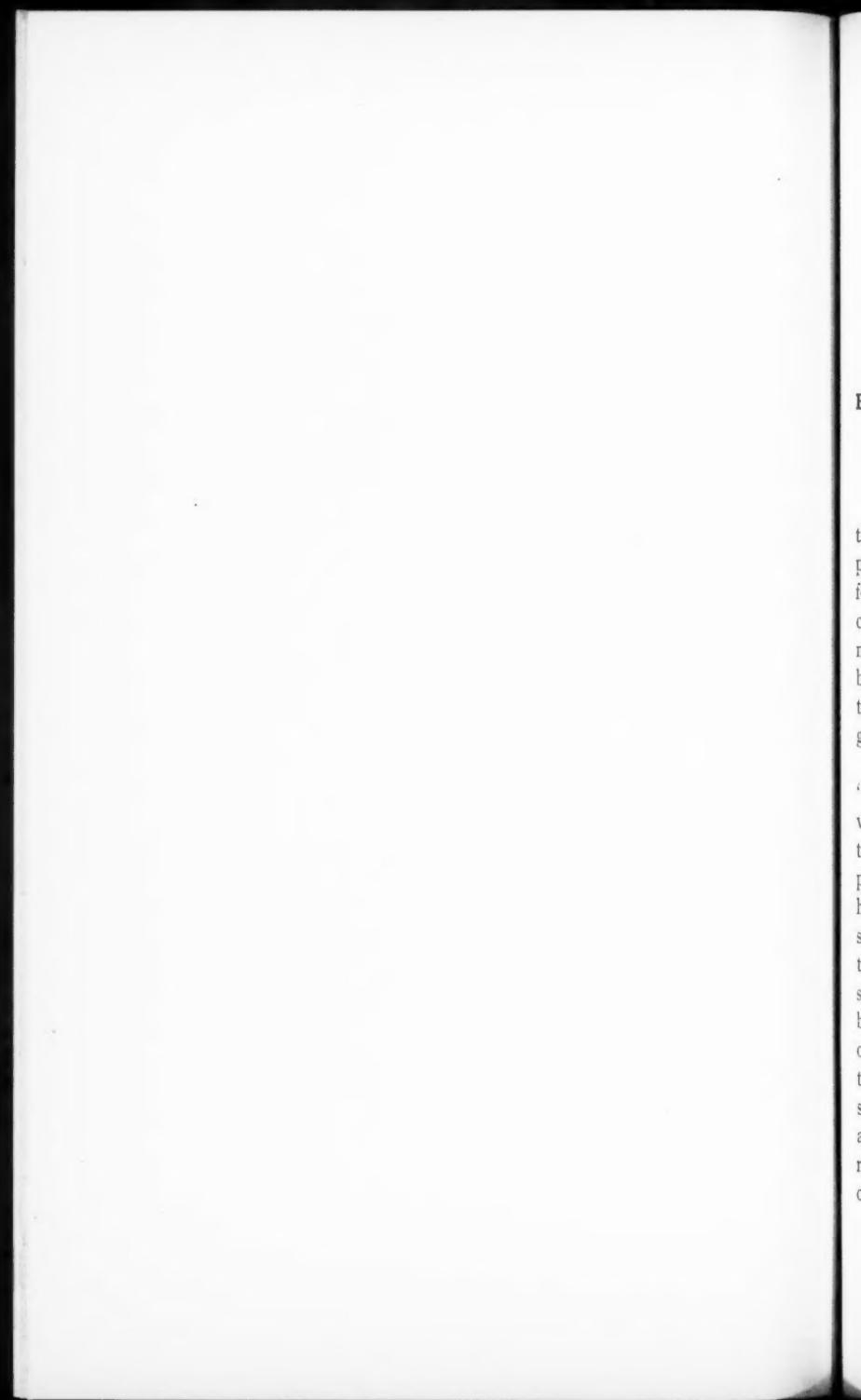
That an irritable colon and a urinary calculus may be coex-

<sup>1</sup> For a description of irritable colon see E. S. Emery, Jr., Disordered Function of the Colon, *Med. Clin. N. Amer.*, 8, 1765, 1924-25.

istent and independent is well illustrated by the third patient's case. Here the right upper abdominal pain and the ribbon-like stools which occurred concomitantly with an emotional storm, preceded the onset of the left-sided pain and persisted after the passage of the stone. Obviously the irritability of the colon, evidenced by right upper quadrant pain is and probably has been entirely independent of the effects of the calculus. The symptomatology produced by multiple intra-abdominal affections is exceedingly bewildering and requires painstaking work and keen discernment to recognize and separate the individual causal components. An ulcer patient who attends the clinic stated that he consulted nine doctors, all of whom recognized the presence of an ulcer, but none succeeded in affording him complete relief in spite of the massive doses of alkalis prescribed. It was not until the patient developed severe colic and gross hematuria that the presence of two independent conditions, nephrolithiasis and peptic ulcer, became apparent. It should be borne in mind, however, that the presence of more than one lesion in a given case is exceptional and that an attempt should always be made to account for symptoms on the basis of a single pathogenetic process.

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## CLINIC OF DR. ISADORE PILOT

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### BRONCHIAL ASTHMA AND VASOMOTOR RHINITIS: REMARKS UPON HYPERSENSITIVENESS AND DE- SENSITIZATION

It is our purpose to emphasize hypersensitivity in three types of patients that present symptoms referable to the respiratory tract. The seasonal cases due to pollens causing hay-fever and asthma will not be considered. In one group are included the patients who suffer with sneezing and profuse watery nasal discharge; in the second, those with typical attacks of bronchial asthma, and in the third, individuals in whom both the nasal symptoms and asthma are combined in varying degrees in the same person.

The patient with sneezing and running nose like that of "hay-fever" is often referred by the otolaryngologist for investigation of the underlying cause. It is of great importance to help this type of patient as a certain number, perhaps 10 per cent., as in hay-fever, develop bronchial asthma. A careful history will often reveal an associated tightness in the chest, with slight wheezing which in the course of time may progress into typical attacks of bronchial asthma. These symptoms representing early manifestations, if alleviated by finding the cause or by other therapeutic procedures, will prevent the development of the less responsive condition of asthma. For these reasons extensive study of these cases to determine the underlying cause should be made. An examination of the nasal mucosa will show a pale, boggy, edematous surface often polypoid. Tissue removed and examined histologically may reveal striking numbers of eosinophilic leukocytes. Ordinary skin tests with proteins in

our experience may give reactions of significance in 30 per cent. of the cases, leaving a considerable number in which the responsible allergens evade us, or possibly etiologic factors other than hypersensitivity may be the basis. By directing our efforts toward finding an offending protein we are doing so because of the better results obtained in such cases. On the other hand, failure to demonstrate a specific protein does not entirely eliminate therapeutic procedures with proteins, as will be later illustrated.

As in bronchial asthma, vasomotor rhinitis may be largely a symptom, rather than a distinct clinical entity, in one patient as an expression of hypersensitivity and in another due to causes at the present time still obscure or not adequately proved. Improvement with calcium administration, quartz light, etc., has not thrown any light upon the etiology as we do not understand fully the changes produced by these agents upon the vegetative nervous system which in these patients have been referred as being in a state of imbalance.

In our cases of bronchial asthma, skin reactions with the dried proteins are obtained in a larger number (40 per cent.) and even higher in children. As in vasomotor rhinitis the asthma may be a symptom due to unknown allergens or to other factors not understood. Most gratifying are the results in children and in cases of short duration. Patients past fifty years of age with asthma of many years' standing are most discouraging. In these persons the added factor of bacterial infection of the bronchi together with permanent tissue changes prevent marked improvement or complete relief. However, all patients should be given the protein tests, for an unexpected offender might be discovered in any patient.

The results of the skin tests in our experience (200 cases), as has been with others, reveal that the inhalants are the most responsible. The foods much less often are at fault and are largely the proteins of egg, wheat, and meats. These foods as well as others are more often responsible in our patients with urticaria and eczema. Bacteria as applied in dried form very seldom give good reactions, but are not excluded unless fresh

sputum cultures are tried intradermally. The history is very important, as it often may lead to the causative agent. However, it is not unusual to find, with skin tests, a protein which could not be elicited in the history although quite apparent even to the patient when discovered. In this connection, from the history we have often predicted orris root, a constituent of face powder, as the cause of the vasomotor rhinitis or asthma, but, as illustrated in the following cases, this substance may turn up unexpectedly.

**Case I.**—A physician suffered with severe sneezing and running nose, often with mild wheezing in chest. In the routine tests he gave a very strong reaction with orris root. When explained it at once occurred to him that his worse days were at the dispensary on women's day in the venereal clinic. It was often necessary for the associates to give the salvarsan injections because of the disturbing rhinitis. Improvement followed by eliminating these days from the clinic, and desensitizing injections with orris root lead to practically complete relief.

**Case II.**—A girl, nine years old, had periodic attacks of asthma and vasomotor rhinitis every one to three weeks. A very strong reaction with orris root was obtained. When found, the mother at once explained the habit of the child playing occasionally with a box containing samples of various cosmetics. The attacks ceased when the box was removed and the patient has now been entirely free from symptoms for three years.

**Case III.**—Girl, twenty-two years old, with vasomotor rhinitis, gave a strong reaction with orris root. She had been avoiding powder but when the cause was brought to her attention she ventured the information that her attacks followed a session at the hair dressing parlor.

At other times it is well to note the observations of the patient, particularly when the ordinary proteins failed to reveal the cause.

**Case IV.**—Boy, fourteen years old, noted that attacks of sneezing were marked when he went out to feed the poultry. The "chicken feed" moistened with saline with the scratch method give an ameboid wheal on the forearm about 5 cm. in diameter.

**Case V.**—A druggist with vasomotor rhinitis and asthma noted that most attacks followed filling prescriptions with "caroid." This substance dissolved in saline applied to arm gave an irregular wheal 4 cm. in diameter. Keeping the "caroid" in a closed place reduced the number of attacks considerably.

A healthy person giving a positive reaction with a substance may develop symptoms when brought in contact with the material. It is also true that a patient may be susceptible to several proteins, although not in contact with these substances. These cases of multiple sensitizations are often difficult to relieve.

**Case VI.**—A physician free from symptoms working with me on the problem of hypersensitiveness in ascariasis gave an excellent skin reaction with extracts of hog and human ascaris. In the course of the problem the round worms were dried and pulverized. At this time he developed marked sneezing, coryza, lacrimation, and soon noted tightness in the chest with distinct wheezing; when the powdered material was sealed in jars the symptoms disappeared.

As has been the experience of others, house dust may give a reaction when ordinary tests fail or in addition to other proteins. Dust from patient's home collected freshly from vacuum cleaner is treated with Coca's solution, filtered through a Berkefeld, and tested by the injection of 0.1 c.c. intradermally in the upper arm. The interpretation of a positive reaction is difficult because of the conglomeration of substances that may be present. In addition, the reaction may not be specific and can sometimes be obtained in some persons free from symptoms. On the other hand, patients may improve markedly when desensitized with these dust extracts, or be entirely relieved when in a new environment.

**Case VII.**—Boy, seven years old, has almost daily attacks of asthma. He gave no marked reactions with the ordinary proteins, but a strong positive with house dust collected in his home. He was removed to the hospital a distance of thirty miles from his home, and that evening and for two weeks following was entirely free from symptoms. He gained weight and strength and was taken home. Attacks returned that night and continued until again removed.

In patients with long-standing asthma, the preparations of autogenous vaccines and filtrates deserve a trial for diagnostic tests and for therapeutic purposes. Our procedure has been to cultivate the washed sputum on human blood agar, aerobically and anaerobically. A mixture of these cultures is prepared as a vaccine. In addition broth containing 1 per cent. of sterile

human blood is inoculated with the washed sputum and incubated forty-eight hours. A filtrate of the blood-broth is prepared by passage through a Berkefeld filter. A filtrate of the sputum is also made in accordance with the suggestions of Wilmer. Skin reactions with these preparations can be obtained in thirty minutes when administered intradermally in some, but not all patients. A reaction in twenty-four hours is common, but may be due to bacterial toxins. A positive reaction would seem to indicate that the bacteria can be sensitizing factors although other proteins may be the primary cause. It is possible that many long-standing cases of asthma become sensitized by the secondary infection of bacteria which may be responsible for the continuance of the asthma even after the original causes are removed. That this may be the mechanism is further suggested by the marked temporary improvement in some patients following the administration of the vaccine, although in our experience permanent relief or cure is not obtained.

In the management of asthmatic patients the removal of responsible proteins may lead to striking results. Such has been the experience in patients sensitized to feathers, orris root, horse dander, and such foods as egg and wheat. In some patients it is advisable to institute a course of desensitization using the protein giving a positive reaction and from the clinical history appears to be the etiologic factor. However, if such a protein is not found, one giving a strong reaction can be used for desensitizing purposes, and in several has given favorable results comparable to specific desensitization.

**Case VIII.**—A girl, aged nine, when first seen, for three years had asthmatic attacks almost daily. A varying amount of generalized eczema co-existed. In repeated skin tests strong reactions were obtained with silk and silk floss. In spite of all precautions possible in avoiding silk neither relief nor improvement was noted. Desensitization with silk was started beginning with 1 : 10,000. Almost immediately the attacks diminished in number and severity, occurring once every month and then less often. When the dose was carried to 1 : 100 the patient was permitted to leave for the South, although from skin tests the patient still gave positive reactions. In a short time the attacks returned almost daily and continued until she returned to the city and desensitizing injections were reinstated. From skin tests it was necessary to return to 1 : 5000. Improvement followed as

in our first experience and continued until injections of 1 : 100 were given when mild attacks appeared. When 1 : 1000 was employed the condition became quiescent and remained thus upon a dilution of 1 : 200 for nine months. It should be noted here that at no time did the skin reaction with the dried protein become negative. The injections were discontinued and in three weeks the attacks returned in severe form for one week. Upon being injected with 1 : 1000 she was again relieved and has been completely free from attacks except for occasional slight wheezing for the past twelve months upon a dilution of 1 : 200. It should be noted that the eczema which at times improved is now very extensive.

Other instances of the effect of desensitization were observed in 4 patients with hay-fever. All had the autumnal type, and gave reactions with the ragweeds. In addition to the hay-fever these persons complained about a vasomotor rhinitis of a lesser degree occurring during the other months of the year. Upon beginning the desensitizing injections with the ragweed pollen in May or June these patients volunteered the information that the non-seasonal sneezing and coryza were absent or markedly improved. All of these patients subsequently had little or no hay-fever. It would seem that the ragweed pollen influenced the seasonal hay-fever specifically and the perennial rhinitis non-specifically. In the same way some of the improvement with vaccines, with injections of house dust, may be rather on a non-specific basis. A tonsillectomy may temporarily lead to improvement which may also be due to absorption of non-specific products from the traumatized area.

Our experience with peptone and tuberculin is limited. With both, only transient improvement occurred; with the former an occasional severe reaction resulted; with the latter, the possible focal reaction in a latent tuberculous lesion discouraged its use.

*x*-Ray irradiation of spleen in 1 patient induced temporary relief; in 2 others no marked effect was noted.

Miss R., thirty-two, developed vasomotor rhinitis when twenty-three years old, in 1919, which continued in spite of treatment and operations until 1924, when she developed a severe asthma. Further operative procedures, autogenous and stock vaccines, bronchoscopy had no influence. Her attacks continued daily, and were very severe, requiring morphin in addition to frequent doses of adrenalin. In repeated tests with proteins a mild reaction was obtained with orris root, 1 : 1000 intradermally. No improve-

ment, however, was obtained with a course of orris root injections. On February 25, 1926 the spleen was irradiated, receiving nearly one-half an erythema dose; on the 26th and 27th the attacks of asthma were fewer; nausea and vomiting developed. The attacks ceased completely until March 11th, when slight wheezing was noted. Polypi were removed from the nose on the 11th and no attacks were noted until April 5th, when she returned with severe recurrences. Owing to the nausea induced by the first treatment, smaller doses of x-ray were administered, this time, however, without effect. It is quite possible that improvement by the first x-ray treatment was due to non-specific protein reaction inasmuch as the large amount of x-ray produced considerable general reaction and a rise of non-protein nitrogen of the blood from 25 to 30 mg. and creatinin from 1 to 1.3 mg.

Ephedrin given by mouth in our experience has been particularly valuable to the patients with very mild attacks, and to those with vasomotor rhinitis complicated by slight wheezing of chest. Some patients feeling an attack coming on state that the ephedrin relieves them when taken early and no severe attack develops. When, however, an attack is well established and of moderate severity the ephedrin gives no relief and hypodermic injections of adrenalin are necessary.

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## DIPHYLLOBOTRIUM LATUM (FISH TAPEWORM) INFESTATION. REPORT OF A CASE OF NATIVE IN- FESTATION IN A CHILD

DURING my service at the Michael Reese Dispensary I was impressed by the number of patients with tapeworm which, upon examination, proved to be more often the fish variety. Further observations in the clinic showed that in each instance the patients with fish tapeworm were all women of Jewish extraction. Several points of clinical interest were noted: First, the absence of an anemia; the absence of eosinophilia, and frequently no symptoms referable to the infestation other than that the patients noticed the segments in the feces. While it is quite possible to harbor the worm for many years it appeared to me that the infestation was recent, particularly when the patients had been in this country as long as twenty or more years, and had passed segments in the few months before first coming to the dispensary. None of these patients were native born so that it could not be stated with certainty that the cases were of native infestation. With a view of determining whether such cases might be native in origin several patients were questioned about eating fish in the raw state. Some admitted liking to eat the fish raw while others denied this, but upon further questioning it was found that the Jewish women prepared the fish by freeing the fleshy portions from bones and chopping these into a minced state. All the patients admitted that they then seasoned the mixture and tasted the raw fish to ascertain the amount of seasoning necessary. My experience in the hospital was similar. At the Lutheran Deaconess Hospital 2 patients with tapeworm were both Jewish women, who sampled the raw fish while preparing it.

It is now recognized that cases of native infestation originate from our fish in the Great Lakes. Lyon<sup>1</sup> reviewed 8 unquestioned

<sup>1</sup> Lyon, M. W.: Native Infestation by Fish Tapeworm, *Diphyllobothrium Latum*, Jour. Amer. Med. Assoc., 86, 264-267, 1926.

cases and added a ninth case in a Jewish boy, four years old. Levy and Pierson<sup>1</sup> in the same year reported another case in a Jewish girl. Both of these children were born in this country, one in Detroit, the other in South Bend, Ind. The blood-picture in these patients as in ours was normal. Calvin's cases<sup>2</sup> were both Jewish children born in Chicago. To this series of undoubted native infestation the following case is added:

H. D., girl, aged seven, Jewish, born in Chicago, a patient of Dr. I. M. Levin, entered the hospital October 3, 1924, for the purpose of removing tapeworm. The treatment had been given three years previously but the head was not obtained. The child had a habit of eating minced fish in raw state when her grandmother was preparing it by chopping. Physical examination revealed no abnormalities except for some tenderness along course of the colon. Blood-count gave 75 per cent. hemoglobin, red blood-cells 4,160,000, leukocytes 14,000; the urine was normal. Stools revealed numerous ova and occasionally segments of *Diphyllobothrium latum*; after the administration of pelletierin tannate she passed about 20 feet of tapeworm. The patient has not had a recurrence up to the present date.

In the cases reported by Lyon, Levy and Pierson, and Calvin no definite history of eating raw fish was recorded. Observations have been made by Dr. Moore and Miss Kaplan in the Out-patient Department of the Research Hospital of the University of Illinois that their patients with fish tapeworm were all Jewish women making a practice of sampling fish in the raw state.

In the following case it is quite possible that reinfection may have been responsible:

Mrs. R. L., thirty-nine, Jewish, patient of Dr. Sahud, born in Russia, was first seen at the Lutheran Deaconess Hospital four years ago. Ten years ago she had had a tapeworm for which she was treated; she passed the fish tapeworm, but it was noted at that time the scolex was not found. For four months stools were examined and no ova demonstrated. She returned to another hospital two years later, when she received treatment and again expelled a worm. She returned on February 10, 1928 with complaints of attacks of weakness, palpitation, dizziness, and diarrhea. These symptoms together with melancholia were noted in the past also when she passed segments. The present symptoms date a few weeks back, when she noticed segments in her stools. Examination of the feces revealed large numbers of the typical ova of *Diphyllobothrium latum*. Blood-count

<sup>1</sup> Levy, D. J., and Pierson, M.: *Jour. Amer. Med. Assoc.*, 87, 848, 1926.

<sup>2</sup> *Jour. Amer. Med. Assoc.*, 78, 84-85, 1922.

showed 80 per cent. hemoglobin; red blood-cells 4,160,000, leukocytes 10,800; small mononuclears 30 per cent., large mononuclears 5 per cent.; polynuclears 63 per cent., eosinophils 2 per cent.; physical examination gave no significant findings. Upon questioning her she admitted that regularly once a week and sometimes oftener she had continued to sample the raw fish while seasoning it in the minced state. Two possibilities present themselves in this case: a recurrence due to failure to remove the scolex or reinfection. She will be observed closely in the future to determine the true situation.

It is quite evident that native infestation with *Diphyllobothrium latum* is occurring about the Great Lakes region. The people affected are Finns and Jews. Among the latter the cases are all in women and children; none in men, although the men eat the same varieties of fish. The history in our patients demonstrated that the fish was habitually ingested in the process of seasoning and still in the raw state, a practice which should be discouraged among Jewish housewives.

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## CLINIC OF DR. LEON UNGER

COOK COUNTY HOSPITAL

### PURPURA HEMORRHAGICA AND ACUTE ENDOCARDITIS

WE present today a most interesting case, one which brings out a good deal of discussion as to the diagnosis. Miss J. C., aged twenty-five, was admitted to the Cook County Hospital, January 19, 1928, with the examining-room diagnosis of "epistaxis." She was taken at once to Ward 21, the Ear, Nose, and Throat Ward, for treatment of a very severe hemorrhage from the nose. She gave the following history:

**Present Complaint.**—Nosebleed, weakness, tendency to bruise easily.

**Onset and Course.**—Patient stated that she had had frequent nosebleed since childhood. These occurred upon very slight trauma to the nose. None of these attacks of epistaxis was severe until this week, when she experienced three seizures of increasing severity. The first one occurred January 14th, and lasted fifteen minutes. The second began the next day and continued six hours. The third one started thirty hours before admission and was so severe that the patient was almost exsanguinated and was brought to the hospital on a stretcher as an emergency.

Patient also complained of weakness, although, according to her statement, she had been strong before these three hemorrhages occurred. All her life she bruised easily. Her gums bled quickly and frequently on slight trauma. However, she gave no history of spontaneous bleeding from mucous membranes, except as already noted. Her last menstrual period kept up for seven days, two days longer than usual. She had no tendency to prolonged bleeding following injuries such as cuts.

**Past History.**—Negative with the exception of the usual mild childhood diseases. She had no recent illnesses. Venereal history was denied and no evidences of such infection were found. Rheumatic history was negative.

**Family History.**—The family history was interesting in that the patient's sister also bruises easily although in good health.

**General.**—Cardiorespiratory history was entirely negative except that dyspnea has been present since the epistaxis began. Gastro-intestinal history has also been negative except that she has been nauseated and has repeatedly vomited blood since her nose began to bleed. Genito-urinary and musculo-cutaneous systems have been negative and her habits are good.

Summarizing her history, we note the severe epistaxis and weakness and the fact that both she and her sister bruise easily. Otherwise, she was in good health up to the past few days.

**Physical Findings.**—On entrance to the hospital the essential points were as follows: Patient was a well-developed, well-nourished, but markedly anemic young woman. She was slightly dyspneic and had a subicteric tint to her skin and sclerae. High-grade pallor of the conjunctivæ and roof of mouth were present.

**Regional Examination.**—Hair, scalp, and ears showed no gross abnormalities. Nostrils appeared normal except for the severe hemorrhage which brought her to the hospital for relief. Her eyes showed no abnormalities save for subicteric tinge of sclera. There were no petechiae here and the pupils were dilated, equal, regular, and reacted to light and accommodation. The mouth showed marked pyorrhea alveolaris and fetor oris and a furred tongue. There were many petechial hemorrhages under the soft palate posteriorly on the right side.

The neck showed no abnormalities and the thyroid was of the usual size, symmetrical, and no nodules were palpated.

**Chest.**—Symmetrical, the excursions full and equal, the skin very pale. There were many petechiae present, especially a cluster of bright red, small hemorrhagic lesions over the right breast. The lungs were essentially negative on admission with the exception of a few scattered râles. The heart was enlarged, the left border being 2 cm. outside the midclavicular line; the right border was substernal. A presystolic thrill and murmur were found at the apex and were not transmitted; the second pulmonic tone was not especially increased in volume. The heart-rate was very rapid and there was no arhythmia.

**Abdomen.**—Normal on admission save for some petechial hemorrhages. The spleen was not palpated at that time.

**Vaginal.**—Examination was not done as the patient was menstruating.

**Extremities.**—Showed many hemorrhagic areas including scattered petechiae and a bluish discoloration, measuring 2 cm. in diameter over the flexor surface of the left forearm, and others about the same size, over both thighs. There was no edema.

*Reflexes.*—Examination of reflexes and lymphatics revealed nothing unusual.

To sum up the physical findings, we note extreme pallor and weakness, slight jaundice, a foul mouth, many large and small hemorrhages under the skin and mucous membranes, severe epistaxis, and an enlarged heart with evidences of mitral stenosis.

The **laboratory findings** revealed the following: Blood: Hemoglobin (F. M.) tests were 45, 43, and 30 on three examinations; erythrocytes were 1,140,000, 1,800,000, and 1,100,000; there were 13,800, 12,200, and 20,500 white blood-corpuscles on three counts. Blood-platelets were exceedingly scarce and the few which were seen were larger than normal. The differential count showed an increased percentage of neutrophilic polymorphonuclear leukocytes and also showed many megaloblasts and normoblasts as well as some poikilocytosis and polychromatophilia. A brilliant cresyl-blue preparation revealed 40 per cent. reticulocytes.

The bleeding time was over seventy-one minutes but the coagulation time was only three minutes; the clot, however, was poorly formed.

The tourniquet test on the arm was followed in a few minutes by profuse petechial rash below the site of application.

Two blood-cultures were negative.

Urine: The urine showed a small amount of albumin on one of three tests, but was otherwise negative. One stool examination showed microscopic blood; this might well have been from the epistaxis and is, therefore, of no clinical importance.

The Wassermann reaction was doubtfully positive and can safely be disregarded. The urea nitrogen was 13 mg. per 100 c.c. of blood, a normal figure.

The important laboratory findings, therefore, included marked reduction in both hemoglobin and red blood-cells, with a color-index above one; a leukocytosis with increased percentage of polynuclear neutrophils, many large and smaller nucleated red blood-corpuscles; poikilocytosis, and polychromatophilia, and a very high percentage of reticulocytes, and, extremely important, a platelet count so low as hardly to be counted, a very

prolonged bleeding time, and a normal coagulation time with soft, non-retractile clot.

**Course.**—Her temperature curve was interesting. On admission it was 100° F., then remained between 99° and 101° F. for the first week; the second and final week it crept up steadily to 103° and 104° F. until it touched 106.6° F. rectally before her demise. Her pulse kept pace and was always rapid; it was never less than 100 and most of the time was between 110 and 130; it reached 145 before the end. Respirations were 20 to 30 the first week, 30 to 45 the second.

Her spleen became palpable before she died.

The course was steadily downward until the patient died, February 3, 1928. During this time she grew weaker and weaker and had one more attack of epistaxis.

**Treatment.**—On admission to the nose and throat ward, the nose was cauterized and the bleeding was stopped. Thromboplastin, opiates, and calcium carbonate were also given. After two days she was transferred to the medical ward for further investigation and treatment.

Two blood-transfusions were done on January 28th and 31st, 400 c.c. whole blood being transferred from her father on each occasion. These gave very little improvement, if any. In addition, subcutaneous saline was injected. The rest of the treatment was symptomatic, except that liver diet was given, with no improvement.

**Discussion.**—Before we give the postmortem results, let us discuss the diagnosis as we faced the situation before death. From the history, physical and laboratory findings, and the course of the case, several clinical entities stand out as possibilities and will be mentioned briefly.

Pernicious anemia suggests itself to us because of the low red count, the high color-index, the megaloblasts and normoblasts, poikilocytosis, and polychromatophilia; also because of the increased percentage of reticulocytes. But against this diagnosis we have the severe hemorrhage, very unusual in pernicious anemia; the markedly prolonged bleeding time; the petechial and ecchymotic lesions; leukocytosis instead of the leu-

kopenia which is the rule in pernicious anemia; the absence of deeply staining macrocytes and microcytes; and, very important, the extremely low platelet count—rarely found in pernicious anemia. Clinically also, there was no history of previous relapses and remissions; the fever was higher than usually occurs in pernicious anemia; and the patient responded neither to transfusions nor to liver diet. On the whole, then, I believe we can safely eliminate pernicious anemia from further discussion.

Aplastic anemia is a disease characterized by signs of injury of all the bone-marrow cells, megakaryocytes, hematoblasts, and myelocytes. Clinically, we find a low red-cell count with low hemoglobin and color-index about 1; the red cells look normal, and there are no evidences of red-cell regeneration, as indicated by nucleated red cells and by increased reticulocytes; nor of red-cell destruction as shown by icterus, etc. Leukopenia with diminution of polynuclears occurs; platelets are decreased in number, and if low enough, hemorrhages result. It can readily be seen, therefore, that in this case only the decreased platelets and hemorrhage and anemia occurred. The findings in this patient of active blood regeneration suffice to rule out aplastic anemia.

Hemophilia is readily ruled out by the following: It occurs only in males, though transmitted through the female; authentic cases in the latter have not been reported. Hemorrhages occur repeatedly beginning at birth or, at least, during infancy. Coagulation time is prolonged and platelets are normal in number. These are sufficient to eliminate hemophilia in this patient.

Acute myelogenous leukemia (myeloblastic leukemia) is suggested by the rapid course, the marked anemia, and the presence of normoblasts and megaloblasts. But it is easily set aside in this case because it is usually accompanied by severe angina and lymphadenopathy and by a large number of premyelocytes in the blood—these latter represent transition stages between myeloblasts and myelocytes.

Acute lymphatic leukemia also has a rapid course with acute onset, hemorrhages, and ulcerative or gangrenous lesions of the mouth. The high lymphocyte percentage makes the diagnosis,

even in the beginning of the disease when the total white count may be normal or subnormal. The chronic leukemias need not be considered here.

Myelophthisic anemia occurs in conditions where normal marrow tissue is crowded out by tumor or tumor-like formations. This causes increased activity of the remaining marrow tissues as shown in the blood. In this condition megakaryocytes are pushed out with the result that blood-platelets are diminished. This type of anemia is found especially where there is metastases to bone, *e. g.*, from hypernephroma or from carcinoma of the thyroid, prostate, etc. No signs of such a process were found in our patient, either before death or after.

We still have two clinical conditions to discuss, both extremely important in this case. These two are acute malignant endocarditis with symptomatic purpura, and purpura hemorrhagica.

In favor of acute malignant endocarditis, we have in this patient the prostration, the high fever, the marked tachycardia, the leukocytosis and, above all, the findings of mitral stenosis by physical examination, and the fatal termination.

The blood findings, with the exception of the very low platelet count, can and do occur frequently in this disease as both blood destruction and blood regeneration are seen. Sepsis is a common cause of severe anemia and streptococci such as can cause acute endocarditis bring on such anemia more often than any other bacteria.

Purpura hemorrhagica (*morbus maculosus werlhofii*) has been defined by Minot as a condition characterized by hemorrhages from mucous membranes, petechiae, or ecchymoses of the skin, a markedly reduced platelet count, a much prolonged bleeding time, and a non-retractile blood-count. The cause of the hemorrhages is probably associated with the deficiency of the blood-platelets with, perhaps, some lesion locally of small blood-vessels. In typical cases the other blood findings are those of posthemorrhagic anemia when hemorrhages occur. Red cells and hemoglobin fall more or less depending on the severity of the hemorrhage; slight poikilocytosis and anisocytosis occur and also

some polychromatophilia; normoblasts are seen at times; reticulated red cells are commonly much increased. Leukocytosis with relative increase in polynuclears is the rule. Bleeding time is much prolonged, but a clot forms within a few minutes. However, this clot is soft, jelly-like and does not retract, and squeezes out a clear serum as a normal clot does.

It can be readily seen that in this patient, all of the above findings were present and present in convincing manner. The hemorrhages were most severe and were associated with the low, almost non-existent number of platelets. The petechiae and ecchymoses were numerous. Added to this finding was the strongly positive constrictor test when petechiae occurred in a few minutes below the site of application of a tourniquet. The bleeding time here (over seventy-one minutes) goes along with the absence of platelets. The history of bruising easily both in the patient and in her sister adds weight to the diagnosis of purpura hemorrhagica.

Our differential diagnosis, then, lies between idiopathic purpura hemorrhagica and malignant endocarditis with secondary purpura. It is customary when a possible primary factor for purpura such as endocarditis is found, to call the purpura secondary or symptomatic and the endocarditis primary and it must be admitted that this order is possibly the correct one in this case. On the other hand, it is also not infrequent, for terminal septic conditions, *e. g.*, acute endocarditis, to develop in the course of true idiopathic purpura hemorrhagica. Favoring this latter conception is the complete absence of any rheumatic history in the patient; this includes also the absence of tonsillitis. Against endocarditis as a primary factor also is to be placed the severe epistaxis, a most unusual condition in endocarditis. Most hemorrhagic lesions in infection of heart valves, are small ones, usually petechial, sometimes ecchymotic—large losses of blood are rare. The history of bruising easily and the presence of this same symptom in the sister also speak strongly for a long continued purpura which for some unknown reason became suddenly severe and death followed. A terminal endocarditis, of course, would have hastened the end. Against endocarditis, also, is the

fact that two blood-cultures were sterile. This, of course, is only one factor and not conclusive when considered by itself.

For the above reasons, then, our antemortem diagnosis was idiopathic purpura hemorrhagica with terminal acute endocarditis.

The most important autopsy findings were as follows: The pericardial sac showed no adhesions and contained about 100 c.c. straw-colored fluid; the pleural cavities each held about 700 to 800 c.c. similar liquid. The tracheobronchial lymph-nodes were slightly enlarged, injected, and anthracotic. The right middle pulmonary lobe had many small, irregular, nodular, reddish-brown masses about 1 cm. in diameter. The bone-marrow of the sternum was very red throughout.

The heart weighed 390 gm., was somewhat enlarged, right side dilated, very flabby throughout. Pulmonary artery  $7\frac{1}{2}$  cm., grossly normal. Tricuspid 11 cm., slightly fibrous thickening of the free margin. Aorta and cusps 6 cm. in circumference, slight atheromatous changes in free portions and commissures slightly obliterated by fibrous adhesions. Mitral valve 7 cm. in circumference, margins and leaflets diffusely thickened by yellow fibrous tissue which extends to the papillary muscle. On the free margin of the mitral leaflet superimposed on the auricular surfaces, were slightly elevated, moderately firm, slightly hyperemic warty-like excrescences. Numerous petechial and purpuric areas were present in the endo- and epi-cardium. The myocardium was very pale brown and flabby and showed no scarring grossly. The coronary orifices were open, and throughout their course were small focal atheromata.

The spleen weighed 535 gm., was rather flabby throughout with a diffuse, purple, mottled surface which showed very slightly elevated, yellow, firm areas which extended into the splenic pulp for a distance of 6 cm. Cut surface very purple, evertting moderately with complete obscuring of normal markings. Splenic vessels not obstructed.

The peripancreatic, hilus, and splenic lymph-glands were enlarged and very red.

The liver weighed 2375 gm., larger than normal and covered by

a smooth, reddish-brown capsule irregularly yellow. The cut surfaces everted slightly and were mottled almost homogeneous red-brown-yellow with obliteration of normal markings.

The inferior vena cava, portal vein, gall-bladder, and biliary passages showed no changes.

The gastro-intestinal tract revealed multiple submucous petechial hemorrhages in the stomach; otherwise grossly normal. The pancreas and duct likewise appeared to be normal.

The kidneys weighed 400 gm. The right one was slightly larger than normal. Capsule stripped with moderate difficulty leaving an extremely pitted, mottled, yellowish-red surface. A few strands of cortical capsular adhesions. Cut section showed the cortex easily differentiated from the medulla and measuring 5 mm. Numerous minute pelvic hemorrhages. Marked hyperemia of pyramids, moderate peripelvic fat. Left kidney similar except that there was a 1-mm. anemic infarct in lower pole of cortex and marked submucous hemorrhages in pelvis.

The ureters and bladder were grossly normal. The uterus was normal in size, smooth surface, slightly boggy. Cut section revealed an intra-uterine blood-clot not adherent to the mucosa. The fallopian tubes were grossly normal.

Histologic sections were made of the kidneys, heart, spleen, and bone-marrow.

The main findings from the pathologic point of view may be briefly summarized from the above:

1. Acute and chronic symptomatic thrombocytopenic purpura with multiple cutaneous, mucous membrane, intra-uterine, and left renal pelvic hemorrhages.
2. Acute verrucose endocarditis engrafted on a chronic stenotic mitral endocarditis.
3. Chronic aortic endocarditis.
4. Chronic fibrous left mural endocarditis.
5. Focal interstitial fibrous myocarditis with one Aschoff body.
6. Acute and chronic focal glomerulonephritis.
7. Anemic infarcts and hyperplasia of spleen.
8. Marked hyperplasia of bone-marrow.

9. Solitary anemia infarct left kidney.
10. Focal bronchopneumonia right middle lobe.
11. Marked cloudy swelling of heart, liver, and kidneys.
12. Bilateral hydrothorax, hydropericardium, and hydroperitoneum.
13. Acute hyperplasia of the tracheobronchial, peri-aortic, peripancreatic, and perigastric lymph-glands.
14. Marked generalized external and visceral anemia.
15. Icterus levis.
16. Focal atheromata coronary arteries and ascending aorta.

The above pathologic report again emphasizes the two conditions, purpura hemorrhagica, as evidenced by the marked hyperplasia of the bone-marrow and spleen, and the numerous hemorrhagic areas; and the endocarditis. The latter, while well marked, was not extreme, and no ulcerations were found. Enough was revealed, of course, to account for the presystolic murmur and thrill made out antemortem.

For the reasons given already, the final diagnosis is as follows: (1) Idiopathic purpura hemorrhagica (*morbus maculosus werlhofii*); (2) terminal acute endocarditis. Acute bronchopneumonia came on just before death and aided in bringing on the fatal outcome.

I am deeply indebted to Drs. McMullen, Tracy, Lyon, Richter, and Liefendahl for valuable assistance.

CLINIC OF DR. LOWELL D. SNORF

WASHINGTON BOULEVARD HOSPITAL

**CHRONIC DIARRHEA. PRESENTATION OF THREE PATIENTS**

In presenting the 3 following case histories I am prompted to do so because of the frequent misunderstandings in both the diagnosis and treatment of diarrhea. It is not possible nor desirable to attempt to cover the entire subject of diarrhea, but to present such phases to you as will have some practical bearing. The term "diarrhea" refers to a stool of mushy and watery consistency and often associated with an increased frequency of defecation. Diarrhea is obviously not a disease entity, but if the stools which tend to be loose are carefully studied much information will be found to aid in proper treatment. Furthermore, a careful study of the history of the patient will frequently reveal a striking picture pointing directly to the diagnosis. As in constipation so in diarrhea, a careful study must be made not only of the stool at the time of examination, but also the condition of the bowel movement from day to day as obtained from the patient's history. Physical and chemical characteristics of the bowel movement are excellent practical criteria of the condition of the colon. Therefore, it is absolutely necessary to include within the history a detailed description of variations of movements. What conception have you of possible variations in a pathologic loose stool? What questions do you ask the patient and what conclusions would you be able to draw or at least speculate upon having secured that information?

Let us refer to the following histories. These reports are from 3 patients who have had chronic diarrhea and have been chosen merely to demonstrate the variations in etiology.

**Case I.**—Mrs. B. W. This patient, a woman aged thirty-four, has complained of symptoms of fulness, epigastric distress, sensation of gas in the abdomen and with a tendency to loose stools at times for ten years. She felt that such foods as coarse vegetables, raw apples, and buttermilk made the intestinal symptoms worse. However, there were times when constipation was present and in consequence cathartics and enemata were resorted to, at first irregularly, but within the past year almost daily.

The gastric distress has not been regularly recurrent after meals; it has not been relieved by adequate alkali or by food. On the other hand, it is much more severe when the stools are mushy and frequent. The distress is aggravated by cold foods and drinks and relieved by warm drinks, is frequently aggravated by a desire for bowel movement and relieved by evacuation of the colon or passing of flatus. She has been on various diets, had tonsillectomy, appendectomy, and ovarectomy, all for the digestive disturbance, but without any result. She was advised to stop cathartics and enemata and following this developed a persistent diarrhea—two to four bowel movements a day and complaining of much pain and colic.

*Examination* reveals a well-nourished woman, general physical findings negative except for a very sensitive abdomen and extremely tender and easily palpable colon. Blood, gastric secretions, urine, and metabolism—all normal.

*Stools* are acid, mushy, and watery, sour odor, and containing much gas, mucus, and undigested food residue. *x-Ray* shows a spastic, rapidly filling colon.

*Treatment* consisted of:

1. Stopping all cathartics and enemata.
2. Soothing low-residue diet, and later bland, and finally soft foods as the colon would tolerate them.
3. Medication—antispasmodics and calcium carbonate to lessen the irritability.

*Comment.*—This history is of interest because it presents a very common symptomatology. To begin with, this patient complains of having had constipation and diarrhea. What evidence is there that this patient was ever constipated? Constipation refers to a bowel movement that is hard and dry or that is unduly delayed. She has had no such stool for years, nor has it ever been delayed for more than thirty-six hours. She has taken a cathartic or enema every day or every other day for a long time to avoid the possibility of a constipated stool. She has always managed to be one or two days ahead of schedule. This procedure was stopped and an ordinary soft diet advised. Persistent mushy and watery stools appear—two or three each day. Examination revealed a highly acid stool with gas, some mucus, and regularly unformed and watery. No blood or pus

observed—condition improving on boiled milk, toast, eggs, and soothing management.

It is obvious that the continuous use of cathartics has produced a marked irritability of the colon in which a perversion of both the motor and secretory function has occurred. With a removal of the cause the function of the intestinal tract returns to normal.

**Case II.**—E. O., aged twenty-six. The patient states that he has had fulness and soreness over the entire abdomen for three years. This distress tends to occur irregularly after meals, at times immediately and again one-half hour after meals, associated with much rumbling and gurgling, eructations, and flatus. He has four to six movements a day which are mushy and explosive. Temporary relief is obtained after evacuation. Hot-water bag relieves but soda and food give no relief. It is always increased by activity, large meals, fatigue, and nervousness. Is nervous, easily fatigued and complains of headache two hours after meals. The appendix has been removed, the tonsils out, and a nasal operation performed all because of his intestinal trouble.

*Examination.*—The heart, lungs, and general examination are essentially negative. However, the abdomen is sensitive and the colon palpable and tender throughout. Stools are mushy, filled with gas, acid in reaction, much vegetable fiber present, no ameba, pus or blood, some mucus. X-Ray reveals an irritability, but no evidence of an organic lesion. Proctoscopic reveals a markedly edematous mucous membrane of the rectum and sigmoid which bleeds easily. No ulcers are noted. Direct smears from the walls of the rectum were negative for ameba and the diplococcus of Bargen.

*Comment.*—Proper consideration of the type of bowel movement should have aided in making a diagnosis very early in the disease. Here the outstanding findings are recurrent attacks of diarrhea, intestinal discomfort, and the peculiar explosive, gassy, sour stools. The diarrhea attacks suggest an irritation which, in the light of the type of stools, definitely stamps it as a fermentative condition. The small intestine is normally a very efficient worker and if the digestive juices are adequate and the irritability not excessive, there will be no undigested foods allowed to pass into the colon to ferment. From where then does the fermentation come? Does this patient have a defective function of the small intestine? Perhaps the findings and subsequent progress of this man will give us the clew.

This patient has taken no cathartics, yet he has frequent bowel movements. This suggests an increased irritability or decreased absorption of the colon. There is no doubt that there is also an increased small intestine activity with a resultant incomplete digestion of the starches. With such an increase in activity the cellulose probably carries starch into the colon. Fermentation is very promptly established with the resultant irritation that follows and a vicious cycle is established. More stimulation from fermentation means more irritation and increased motor activity of the intestine and consequently more starches in the colon.

Excessive roughage alone did not produce the irritation in this instance. All starches, sugar, etc., had to be removed before the colon became quiet and the stools normal. Nor was there a deficiency in the pancreatic or intestinal juices since the starches, milk, and sugar were added finally with impunity after the irritability ceased. It would seem, therefore, that here the irritation from the by-products of fermentation was the chief and primary factor.

It must be remembered, however, that in this type of condition there will be found great variations. For instance, one patient may present a history very similar to Case II with the exception that the error in diet is quite pronounced. Much roughage, raw fruit, candy, bran, buttermilk, etc., have been indulged in from time to time either because of fancy or misapprehension. The fermentative condition is present, but will promptly clear up by eliminating the roughage.

There are variations up to the more severe type similar to the intestinal fermentative dyspepsia described many years ago by Schmit. The latter require infinitely more care in differential diagnosis, but this can be done by test diet and proper evaluation of all chemical tests. Here the high protein, low carbohydrate diet will give very prompt relief. The roughage has much less a place in the etiology while the dysfunction of the small intestine seems to be the big factor.

Cases I and II are very similar in the final analysis. In one irritation was produced by prolonged cathartics, while the

other bowel was excessively active because of irritation from food and by-products of fermentation. In either event the condition is one of irritable colon. However, it is quite obvious that the specific etiologic factor must be recognized in order to obtain the proper result in treatment.

**Case III.**—J. M., aged sixteen. This young man's trouble began rather suddenly one and a half years ago with a profuse, watery, blood-tinged diarrhea. It is likely that there was temperature because of feeling chilly at times. Examination of stools was made, but no parasites are said to have been found. He was placed on a milk and cooked cereal diet. There seemed to have been temporary relief but after six weeks there was more or less constant watery and mush diarrhea continuing for a year. Every month or six weeks there would be a week or more during which time flecks of blood would be noted and some pain complained of over the lower left quadrant of the abdomen. The patient was then placed on a high protein carbohydrate-free diet with formed stools following very promptly. It was assumed that that was the end of the trouble. After six weeks or two months there occurred the bleeding and diarrhea again which was similar to the early attacks. The patient then discouraged returned to a general high starch diet and promptly developed a severe diarrhea. At that time he comes to the hospital complaining of diarrhea, gas in bowel movement, much weakness and easily fatigued.

*Examination* reveals a young man, quite emaciated, skin dry and pale, hemoglobin 68 per cent. All organs are negative except for moderate tenderness of the abdomen, especially over the lower left quadrant. Stools are watery, contain much mucus, alkaline reaction, flecks of blood and pus, and much gas is formed when allowed to incubate. The gas bacillus of Welch was isolated in large numbers. *x-Ray* suggests a chronic ulcerative process because of marginal feathering, pointed out by several authors, and marked spasm. Proctoscopic examination revealed a mucous membrane which bleeds easily, is congested at rectum, but in vault of rectum and in sigmoid are found numerous small ulcers, pin-head to millimeter in size. Warm stage examination was carried out on four different specimens before the *Amoeba histolytica* was found. Scrapings of the ulcer base also revealed the organism.

Before beginning the anti-amebic therapy he was placed on a high protein, low carbohydrate diet and with almost complete disappearance of the diarrhea. This was done merely to show the effect diet would have on a diarrhea associated with a colon overrun by Welch bacilli. Specific treatment was then instituted, with a complete disappearance of all ulcers.

**Comment.**—This patient presents problems not emphasized in the other two.

*Diarrhea* present as long as in this patient should be very significant. It must be looked upon as potentially organic until

proved otherwise, and after a careful stool analysis and proctoscopic examination the problem is often not very difficult to decide. Blood may be noted in the stool occasionally and not refer to anything more than hemorrhoids or congestion of mucous membranes in the rectum, but when present at intervals as dark flecks and bright at times, mixed with the stool, it is very likely to be due to ulceration. The true pathology was overlooked because of failure to appreciate the possibilities. Proctoscopic and sigmoidoscopic examinations should be done routinely in all chronic intestinal disturbances. Carcinoma may be beyond the examining finger, but within easy reach of the scope. The  $x$ -ray, on the other hand, may reveal nothing in rectum or distal position of the sigmoid just where the early chronic ulcerative processes may occur.

The temporary good result in this patient obtained from the high protein diet, as compared with high carbohydrate diet, is of passing interest. Rule of thumb treatment will not work. Careful examination of feces should have revealed the type of diet and treatment required, but instead months passed before a change was offered.

**Discussion.**—Diarrhea is due usually to trouble in the colon rather than the small intestine. Certain findings suggest that the small intestine is involved either primarily or in association with the colon, namely, the stools are acid in reaction, contain much poorly digested food residue, mucus is intimately mixed with the stool, unreduced bile-pigment is present, and there is excessive loss in weight. When the colon is involved especially in organic disease, the stools are alkaline, mucus is separate especially if in the lower bowel, and bile is reduced. Stools may be acid in reaction in which both the colon and small intestine are involved as in the presence of such functional disorders, as irritable bowel and other motor and secretory perversions. Except in the presence of organic disturbances of the small intestine the diet management is directed largely to the large bowel. Irritants as cathartics, irritating foods, or psychic factors and emotional disturbances which produce an influence on the intestine may, if not controlled, develop into a true anatomic lesion.

Chronic diarrhea may be the presenting symptom, however, where the primary trouble lies elsewhere than in the intestinal tract. Toxic goiter, tuberculosis, and Addison's disease are notable examples.

Anatomic disturbances of the colon are due to non-specific ulcerative colitis and sigmoiditis, amebic dysentery, tuberculosis, enterocolitis, diverticulitis, carcinoma, syphilis, sprue, and pel-lagra. These diseases can only be differentiated by careful and painstaking examination.

**Treatment.**—The management of chronic diarrhea must be dependent upon the etiology and underlying pathology. In general the most important factors are diet, controlling the patient's habits, and medication.

The patient is put to bed for the necessary time. During the time of treatment opportunity is given for further study to eliminate such underlying pathology as gall-bladder disease, appendiceal affections, and organic disturbances in the colon itself. Rest, sleep, and proper mental adjustment are necessary to secure the best results. Heat is applied to the abdomen, small enemas are given when the stool is hard and dry, but caution must be used not to use water injections too frequently or in large amounts, merely because of lack of bowel movement. A daily digital examination of the rectum will keep the physician informed as to whether a water or oil retention enema is necessary.

Such foods are chosen as will produce the minimum amount of stimulation to the colon. The bland foods as boiled milk, cooked cereal, toasted white bread, eggs, and rice, are reasonably non-irritating. Often in the first few days a diet of gruel and boiled milk must be followed. Gradually additions are made to the diet with as much care and precision as in the prescribing of medicine. Cooked vegetables are added, one at a time, preferably puréed, and then cooked fruits. The raw vegetables and fruits are cautiously added only after it is evident that the intestine will tolerate all the cooked fruit and cooked vegetables necessary. The time required may be from a week to a month or more. Careful instruction and follow-up care is necessary to secure a lasting result.

Medication is usually relatively simple. Opium has been used for many years, but should only be resorted to after other drugs have been tried unsuccessfully or when the diarrhea is producing great dehydration.

Calcium lactate or calcium carbonate with bismuth subcarbonate is given in 20-grain doses three to five times daily. This also tends to decrease the peristaltic activity of the intestinal tract. Tincture of belladonna with small doses of sodium bromid should be used in presence of colic.

The ulcerative lesions demand the above therapy plus such specific treatment as indicated in the particular case.

**Résumé.**—1. Every patient with chronic diarrhea should have:

- (a) Careful history and physical examination.
- (b) Careful microscopic study made of stools.
- (c) Proctoscopic examination.

2. Treatment will depend entirely upon results of these examinations.

Diet management will not be satisfactory if not carefully controlled by frequent stool examination.

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## CLINIC OF DR. JACOB MEYER

MICHAEL REESE HOSPITAL

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### RENAL GLYCOSURIA AND DUODENAL ULCER

RENAL glycosuria is a term applied to patients showing a normal concentration of blood-sugar with glycosuria which is unaffected by the diet. This condition is said to be hereditary or familial. The patients show no symptoms of diabetes mellitus, such as hunger and thirst. For a definite diagnosis of this condition it is necessary to observe the patient over a period of years. The blood-sugar tolerance tests are normal. Joslin<sup>1</sup> advises against the diagnosis of renal glycosuria, saying it leads to carelessness on the part of the patient and the doctor. He mentions Jacobsen and Ohler as agreeing that certain cases reported as renal glycosuria, in later years developed true diabetes. The exact mechanism involved in renal glycosuria is not clear. It may be well to recall that when the concentration of the blood-sugar rises above 160 to 180 mg., sugar appears in the urine. This is spoken of as the normal sugar threshold. In renal glycosuria, glucose appears in the urine, with a blood concentration of 100 mg. or less. One may speak of it, then, as a lowering of the normal sugar threshold, or again as a functional glycosuria.

The cases that I wish to present to you are of interest because they are two brothers, both of whom were admitted because of duodenal ulcer. Although each was aware of the presence of sugar in the urine, this was not a part of the complaint, the nature of the glycosuria being determined only after study.

**Case I.**—Jacob L. had been under observation at Michael Reese Hospital from January 21 to February 19, 1927 and had been discharged with the diagnosis: Gastric ulcer (?), duodenal ulcer, and mild diabetes. A typical ulcer story of five years' duration had been obtained at that time. He had

been operated at a well-known clinic in 1924, a gastro-enterostomy having been performed. Relief of gastric discomfort was present for one and a half years. His symptoms then recurred. This caused him to enter the hospital. The roentgenologic report (Dr. Arens) in January, 1927 was: "Examination discloses a postoperative gastro-enterostomy with tenderness directly over the stoma. No defect was noted. There was considerable fluid in the stomach. The enterostomy opening functions slowly, the barium not passing through for a short period of time. The bulbus duodeni is defective, remaining so throughout the examination. The films confirm the fluoroscopic examination."

The urine on admission at this time was positive for sugar and the blood-sugar was 100 mg. The physician in charge of the service considered the patient as a mild diabetic and he was placed on a restricted carbohydrate diet. Thus the patient with a blood-sugar of 100 mg. on a diet of carbohydrate 30, protein 40, fat 100, showed 4 gm. of glucose in twenty-four hours.

	Date:	1/27/27	1/29	1/30	1/31	2/1	2/2	2/3
<i>Urine:</i>								
Amount . . . .	600 c.c.	540 c.c.	840 c.c.	600 c.c.	510 c.c.	540 c.c.	540 c.c.	
Sp. grav. . . .	1026	1025	1022	1022	1026	1023	1021	
Sugar . . . .	Hg.	Hg.	0	0	3 gm.	8 gm.	4 gm.	
Acetone . . . .	+	+	trace	++	++	+		
<i>Diet:</i>								
Protein . . . .	40 gm.	45 gm.						
Fat . . . .	100	100	100	105	125	125		
Carb. . . .	30	35	35	40	40	45		
<i>Blood-sugar:</i>								
	100	100						
<i>Insulin:</i>			5 units					

Sugar persisted for a few days and then disappeared with the discontinuance of insulin. The patient was discharged with a diet of protein 58, carbohydrate 60, fat 147. No sugar was in the urine on the day of discharge.

On October 15, 1927 he was admitted to the surgical service. Ten days previously the patient developed severe pain in the epigastrium. Two days later pain recurred much more severely. The patient was nauseated but did not vomit. Physical examination revealed an acutely sick man, temperature 99.2° F., pulse 104, respirations 54. The abdomen was rigid and extremely tender. The urine on admission showed sugar +++, acetone ++ and a trace of diacetic acid. The *blood-chemistry* showed *blood-sugar* 96 mg. per 100 c.c. and non-protein nitrogen 34 mg.

A diagnosis of ruptured gastric ulcer was made, and patient

was operated. Operation revealed a perforated ulcer just beyond the pyloric ring on the anterior surface of the duodenum.

The next morning, October 16, 1927, the urine contained sugar +++, acetone +++, no diacetic acid.

Five units of insulin was given twice daily. Blood-sugar 100 mg. Sugar varying from 18 gm. to 2 gm. was present in the urine daily. Acetone was also present. Fluids were given, glucose and soda bicarbonate per rectum. On October 20, 1927, the patient was placed on a gastro-intestinal diet. Milk and cream, 2 ounces every hour, was also given. Insulin was discontinued.

*Sugar Tolerance Test November 11, 1927*

Blood-sugar at 7 A. M. =	80	mg.
85 gm. glucose then given at 7.30 A. M.—blood-sugar	148	
8.00 A. M.—	"	163
8.30 A. M.—	"	133
10.00 A. M.—	"	76

On discharge from the hospital, patient was receiving milk and cream every hour, and five feedings, such as cereal, soft-boiled egg, custard, vegetable purée, etc. On this diet he showed 5 gm. of sugar in the urine.

**Case II.**—Harry L. was admitted to the surgical service on October 26, 1927, complaining of pain and a hernia in the epigastrium. Fifteen months previously the patient was operated for "gastric ulcer" at a local hospital, a gastro-enterostomy being performed. Eight months prior to the operation an insurance company physician had told him that he had "diabetes." He now complains of pain in the epigastrium which comes on from two to three hours after meals. He is also uncomfortable because of the bulging in the epigastrium and wishes this repaired.

Roentgenologic examination (Dr. Arens): "Films disclose a postoperative gastro-enterostomy. It is difficult to palpate the stoma for tenderness, tenderness being present also over the postoperative scar. The prepyloric area is defective and tender on palpation. The findings of a gastrojejunal ulcer are not definite."

The urine on admission was positive for sugar, and the blood-sugar was 79 mg. I was asked to see this patient and expressed the opinion that this patient was probably a case of renal glycosuria, in view of the low blood-sugar, the glycosuria, the absence of diabetic symptoms and because his brother was in the ward with an almost identical picture. The blood-sugar tolerance test showed a deviation from the normal.

November 2, 1927: Blood-sugar = 82 mg.

85 gm. given and $\frac{1}{2}$ hour later—blood-sugar	174 mg.
1 hour	256 "
2 hours	176 "

Blood-sugar repeated on November 21, 1927.

25 gm. of glucose	105 mg.
$\frac{1}{2}$ hour later	170 "
1 hour	176 "
$1\frac{1}{2}$ hours	143 "

The patient was operated on November 3, 1927, for the epigastric hernia and in addition to numerous adhesions between the liver and stomach, colon, and stomach and abdominal wall, a gastrojejunal ulcer was noted. The patient continued to show sugar in the urine during the entire stay in the hospital. Blood-sugar remained normal. The patient has been under observation for his gastrojejunal ulcer and is to be operated for same. Though still showing sugar in the urine, his blood-sugar remains normal.

The 2 cases presented fulfil the requirements for the diagnosis of renal glycosuria. It is well to bear in mind, however, that peptic ulcers are often associated with low-grade infections of the adjacent organs such as the liver and pancreas. This is particularly true of penetrating lesions involving the posterior wall. The question might well be raised as to whether or not the glycosuria was due to an associated infection of the pancreas. My impression is that in glycosuria associated with infection, the blood-sugar concentration is increased and progresses with the severity of the infection.

Attention is also directed to the ketonuria present in the first case. Acetone was present on the first admission and again at the second admission. This would very readily lead to the diagnosis of diabetes mellitus. However, ketosis has been previously observed in renal glycosuria. Allen, Wishart, and Smith<sup>2</sup> reported 3 cases of renal glycosuria with ketonuria. Patterson and also Tausig<sup>3</sup> reported a case. In our first patient the explanation of the acetonuria may be due to the acute local peritonitis when rupture occurred.

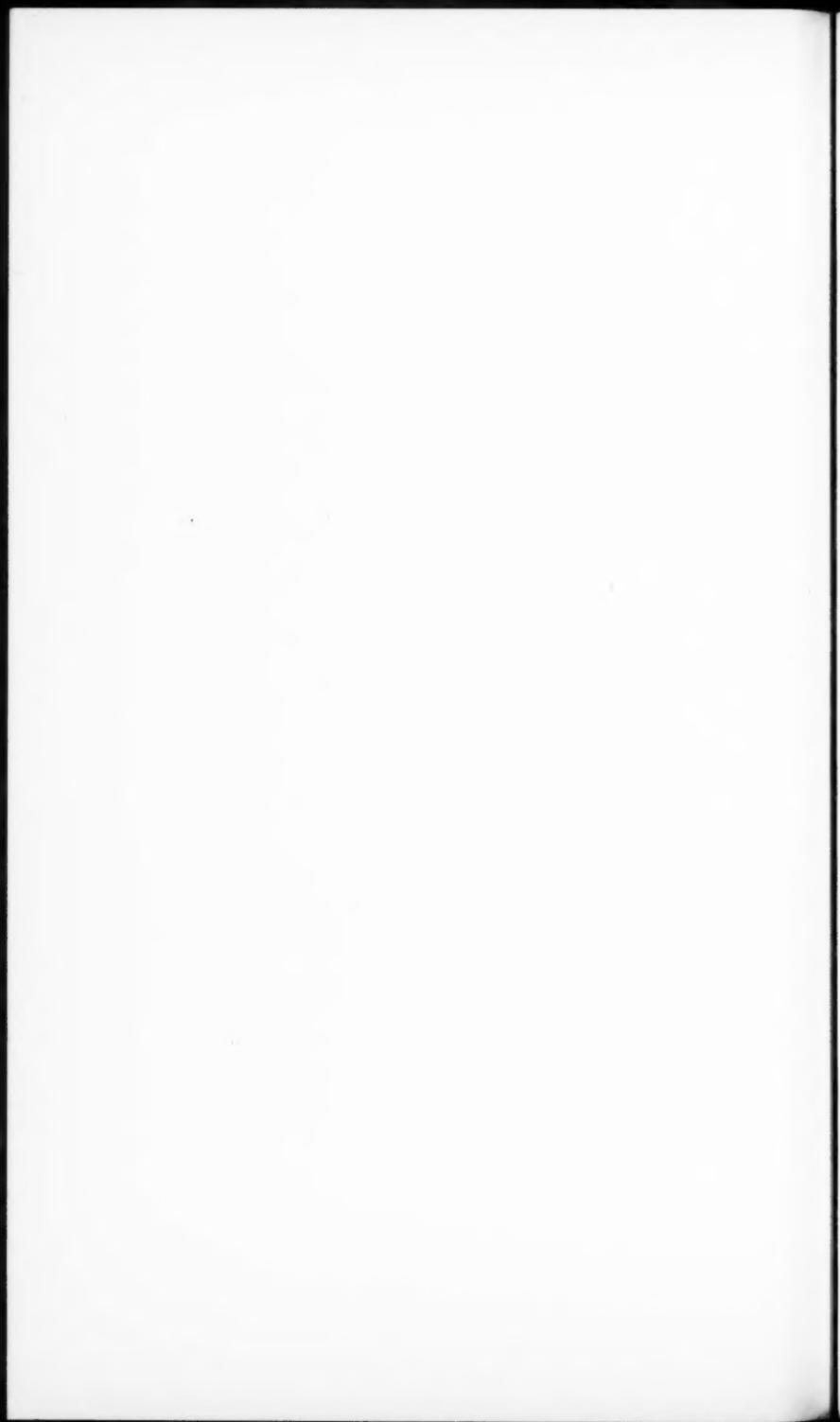
In the treatment of the first case, J. L., we assumed that he was a true diabetic. Fluids were forced postoperative. Glucose and sodium bicarbonate were given per rectum, and 10 units of insulin daily. Later when we felt more certain of the patient,

the insulin was reduced and then discontinued. In the case of the brother, H. L., the postoperative treatment was simplified. I did not recommend insulin and general ordinary postoperative routine was used.

Lest some may misunderstand, I wish to caution against such procedure in the postoperative treatment of true diabetes mellitus. After an operation on a true diabetes mellitus, the internist should give strictest attention to the patient for twenty-four to forty-eight hours. Fluids should be forced, by rectum, subcutaneous, or intravenous method. Glucose 250 c.c. of a 10 per cent. solution given intravenously with 10 units of insulin is a good routine procedure. Orange-juice with sugar by mouth, if the operation permits, is also valuable. The blood-sugar should be determined as often as possible, daily or more frequently, and the patient's urine observed in similar fashion. It is well to consider a postoperative diabetic as an impending coma. The further detailed treatment will necessarily vary with the individual case.

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## MASSIVE COLLAPSE OF THE LUNG

H. L., primipara, aged twenty-three, was admitted to the obstetric service of Dr. J. Baer, and operated by cesarean section under ether-ethylene anesthesia, November 2, 1927, because of generally contracted pelvis, full-term cephalic presentation with marked overriding. Membranes were ruptured with discharge of meconium, pulse going up to 180. The patient left the operating-room in good condition. The pulse of patient on arriving in the ward, according to nurse's chart, was 100 to 88, respirations 18 to 20. Early on the morning of November 3, 1927, the patient was reported as sleeping, dozing with some hoarseness. At 7.00 a. m., November 3, 1927, patient was turned on the left side by nurse, who reported patient had mucus in the throat and complained of pain in the left arm and shoulder. Pulse was 118, respirations 24. I was asked to see patient about three hours later.

Physical examination revealed a young woman, comfortable, moderate cyanosis, and a mucous rattle was heard on approaching the patient. The chest was hyperresonant anteriorly and posteriorly, with numerous diffuse râles over the entire chest; no evidence of consolidation. The heart-tones were rapid and good quality. No definite cardiac displacement was demonstrable but it was noted that the normal liver dulness was displaced by hyper-resonance. A tentative diagnosis of postoperative massive collapse of the lung was suggested.

The recommendations were as follows: Morphin sulphate for sleep to be given as indicated; tincture digitalis, 15 drops three times a day; move the patient frequently from side to side. The pulse continued at 112 to 114 per minute until the following morning when the pulse suddenly increased to 160 to 172 per minute. Respiration was 24. Cyanosis was more intense. I now saw the patient and made the following notations: "Cyanosis marked, pulse rapid, poor quality. Heart is displaced to the right side. This is demonstrated by percussion and the heart-tones are very definite to the right. The breath-sounds are loud tubular in quality over the right lower lobe. The same is heard at the left base. Hyperresonance is present over the right lower lobe, posteriorly. There is no displacement of the liver or spleen. The diagnosis of massive collapse of the right lung was now definite." An x-ray examination showed on November 4, 1927, "a clouding of the right lung field with almost complete retrac-

tion of the heart and mediastinum to this side. The left lung field is emphysematous. The appearance is that of massive collapse of the lung."

The patient was observed daily until discharge. Physical findings of consolidation were present in the right lower lobe. Thus on November 6th, dulness in the right lower lobe, breath-sounds are heard on forced breathing; tubular breathing is also heard. Anteriorly there is hyperresonance.

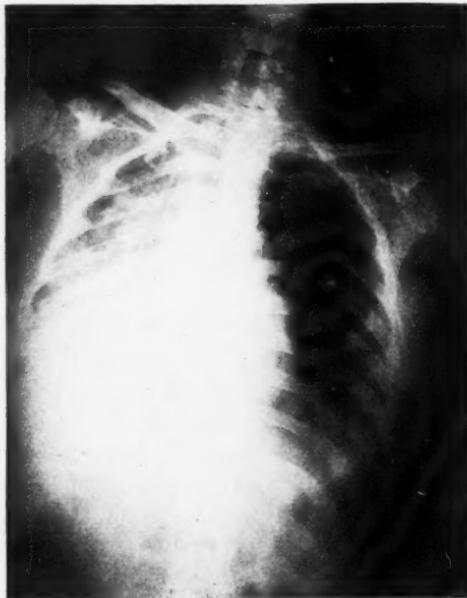


Fig. 42.—Showing collapse of right lung and displacement of heart.

The heart was noted to gradually return to normal position with corresponding drop in pulse-rate.

The subsequent course was uneventful. *x*-Ray films were taken to continue the study. Thus on November 7, 1927, Dr. Arens reported: "The films now reveal a marked increase of air in the right lung with the heart and mediastinum almost back to normal as compared with previous examinations." One

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week later, November 14, 1927, "The films which previously disclosed a massive collapse now disclose both lung fields within normal except for a small area of cloudiness extending from the right hilus down towards the base, probably a pneumonic patch. Both lung fields are of equal density. The heart and mediastinum are in their normal position."

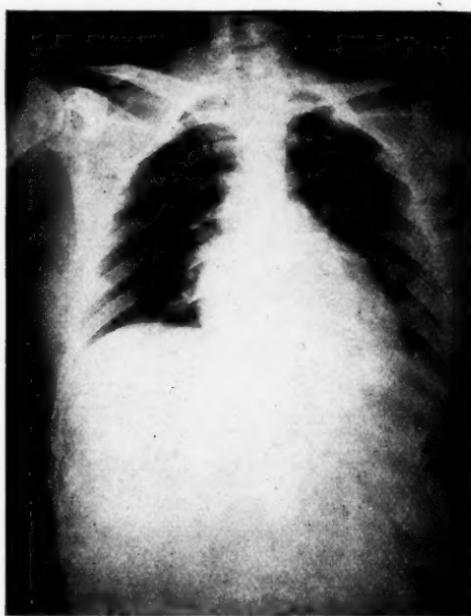


Fig. 43.—Return to normal.

The patient was discharged in normal state and was seen one month later in the Out-patient Department with no evidence of physical change.

I will refer those of you who wish to read the subject of massive collapse, to the excellent article by Sir John Rose Bradford.<sup>1</sup> It is to him that we are indebted for the recognition of this condition in civilian practice.

<sup>1</sup> Bradford, Sir John Rose: Massive Collapse of the Lung. Oxford Medicine, vol. ii.

I should like to direct your attention to the early recognition of this condition in this case, and the gradual development into a complete right-sided collapse. Bradford mentions a case of a soldier, collapse occurring fourteen to sixteen hours after the time of wounding—"the soldier walked four miles after being wounded." Our patient developed symptoms the day after operation, and in forty-eight hours the physical findings of massive collapse were complete.

The tachycardia is also of interest. Bradford does not speak of this condition and as there is no temperature, the tachycardia cannot be ascribed to infection as a cause. I attribute the tachycardia to a mechanical embarrassment of the heart.

Displacement of the heart and tachycardia following an operation may be interpreted as an acute cardiac dilatation. This is often the diagnosis. Especially is this so because of the presence of mucous râles, leading to the erroneous conclusion of pulmonary edema. Attention is directed to the importance of the *absence* of orthopnea in massive collapse. Characteristic of massive collapse are the physical findings of displacement of the heart, to the side of the collapsed lung, the high diaphragm, the findings of consolidation of varying degree over the area of collapse, the sudden or gradual alteration in the physical signs, and the relative comfort of the patient.

The prognosis is, on the whole, good. Patients are sometimes unaware of the condition and physicians have often overlooked it. Those cases due to aspiration of mucus secretion, which block a bronchus and thus lead to collapse may subsequently develop a lung abscess.

The treatment is simple rest, cardiac stimulation if required, and recently, frequent turning of the patient from side to side has been recommended.

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CLINIC OF DRs. SIDNEY A. PORTIS AND  
SAMUEL J. HOFFMAN

COOK COUNTY HOSPITAL

**CASE I. CARCINOMA SIMPLEX OF STOMACH AND**

**CARCINOMATOUS LYMPHANGITIS OF LIVER**

**CASE II. ACUTE HEMORRHAGIC PANCREATITIS WITH  
SECONDARY CYST FORMATION**

**CASE III. SUBACUTE BACTERIAL ENDOCARDITIS**

WE are presenting 3 unusual case histories this morning with necropsy findings because they offer interesting points in discussion of the diagnoses.

**CARCINOMA SIMPLEX OF STOMACH AND CARCINOMATOUS  
LYMPHANGITIS OF LIVER**

**Case I.**—The first case is that of a male negro, age forty-five years, who entered the Medical Service, Ward 54, of the Cook County Hospital with the complaint of pain in the abdomen, vomiting, constipation, and loss of weight, all of six months' duration. The patient was feeling well until about six months ago when he noted pain in the epigastrium which was dull in type and appeared two to three hours after eating, and was relieved by vomiting. The pain had been getting gradually more severe until the time of admission. He was also awakened at night with pain which was relieved by vomiting. The pain was unaffected by bowel movement. There was no gross blood in the vomitus, although there was definite evidence at all times in the latter few months of retention of more than seven hours' duration. He had lost between 20 and 25 pounds during this period. Constipation was relieved by saline cathartics.

The past history revealed the fact that he had pneumonia at sixteen, chancres and gonorrhea at twenty years of age, no surgical operations.

The family history was essentially negative. There were no pregnancies.

Physical examination at time of admission revealed a well-nourished, colored adult male, about forty-five years of age, with normal temperature, pulse, and respiration, and a blood-pressure of 90/55. The scalp was essentially negative, as were the ears, nose, and mouth. The pupils were irregular, did not react to light, but to accommodation. The lungs were essentially negative and there were no murmurs or irregularities of the heart. There was very definite tenderness in the epigastrium just above and to the right of the umbilicus. However, there were no definite masses palpable. The liver

edge was sharp and firm, not irregular, and palpable 2 cm. below the costal arch in the midclavicular line. The abdomen was otherwise negative. The knee-jerks were absent, but the Achilles jerks were present. There were no other abnormal reflexes. There was slight generalized lymphadenopathy including the epitrochlear glands.

The stomach analysis revealed no free acid, a total of 40 with the Ewald meal, and on the motor meal there was distinct evidence of retention with no free acid and a total of 75. There was no blood in either specimen. Lactic acid was absent. Boas-Oppler bacilli were not found.

Repeated stool examinations showed only a faintly positive benzidin.



Fig. 44.

The urine had only a trace of albumin. There was no sugar, diacetic acid, or acetone. There was an occasional red blood-cell and a few pus-cells. Casts were absent.

The blood-count revealed 75 per cent. hemoglobin, 3,850,000 red cells and 8300 white cells with a normal differential. The blood chemistry findings were normal except for an icterus index of 8, and a CO<sub>2</sub> combining-power of 60 per cent. The Wassermann test on the blood was negative. A spinal puncture was not done because the patient was too ill.

The fluoroscopic x-ray examination revealed a distinct and gradual narrowing of the stomach beginning in the pars media and extending up to the pylorus. There was no marked irregularity of either gastric curvature. However, it had lost its smooth appearance in the involved region compared to the uninvolved region. The plates confirmed the fluoroscopic examination, showing a definite deformity progressing toward the pylorus (Figs. 44, 45).

During the first few days of observation in the medical ward the patient became gradually worse, with continuous vomiting, and surgical intervention was deemed advisable. He was operated on by Dr. Paul Oliver of the Surgical Service, who found at operation the distal 3 inches of the stomach wall thickened, firm, pliable, but not nodular in its distal third. There were a few omental tags involving the greater curvature side of the stomach which contained firm, web-like nodules. The periaortic, pancreatic, and peribiliary lymph-glands were firm and slightly

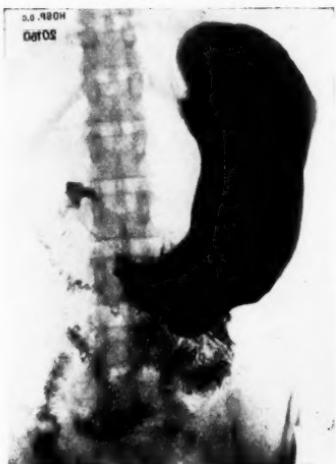


Fig. 45.

enlarged, but not "stony hard." The liver was firm, smooth, definitely enlarged, and had a red to brown mottling, but no evidence of gross metastases. A gastro-enterostomy was done. Sections of the liver and stomach were taken for examination.

This case is interesting because it offers three possible diagnoses preoperatively—first, a carcinoma planum of the stomach; second, syphilis of the stomach, and third, plastic linitis. Carcinoma of the stomach from these clinical and laboratory findings cannot be ruled out. Syphilis of the stomach because of the clinical picture associated with a definite history of lues and sug-

gestive fluoroscopic and radiographic findings should be seriously considered even in the absence of a positive Wassermann. Linitis plastica must be included because the clinical picture and radiographic findings may be associated with this condition. At operation it was difficult to determine the exact nature of the lesion and for that reason sections were taken.

The patient remained on the Surgical Service, continued to fail, and finally died eleven days after operation. At necropsy there was found an infiltrating carcinoma of the gastric wall at the pars pylorica with slight ulceration of the mucosa and partial stenosis of the pylorus; diffuse carcinomatous lymphangitis of the liver; carcinomatous metastases of the periportal, peribiliary, and coronary lymph-glands; acute focal fibrinopurulent peritonitis engrafted on a hydroperitoneum; cloudy swelling of the parenchymatous organs; scarring of the myocardium; slight atheroma of the mitral leaflets; slight atheroma of the aorta; calcified nodules of the pulmonary apices and calcification of the tracheobronchial lymph-glands; subacute tracheobronchitis; recent gastrojejunostomy; recent supra-umbilical laparotomy wound; multiple small infarcts of the spleen; right lower lobe pneumonia; chronic passive hyperemia of the abdominal viscera; chronic cholecystitis; benign hypertrophy of the prostate gland; generalized superficial adenopathy; generalized emaciation, dehydration, and anemia; unequal pupils.

Microscopic examination of the sections from the stomach and liver revealed carcinoma simplex of the stomach and carcinomatous lymphangitis of the liver.

#### **ACUTE HEMORRHAGIC PANCREATITIS WITH SECONDARY CYST FORMATION**

**Case II.**—The second case record is that of a white male, thirty-eight years of age, who entered the Medical Service with the history of being comparatively well up to five months before entrance, at which time he was suddenly seized with severe abdominal pain, pointing to the epigastrium, which was sharp, stab-like in character, and radiated to the left shoulder, so agonizing and excruciating that the patient was "doubled up." The pain, although milder in type, lasted about one week and confined the patient to bed. He said that he was never jaundiced during or after the attack. He further stated that a dull pain followed this acute attack and had been more or less constant up to the time of admission. It was aggravated by the

intake of food and not relieved by alkalies. He had several similar acute attacks, the last one being two weeks before admission. With the last attack he noted a swelling in the epigastrium which progressively became larger. Vomiting commenced two weeks before admission and became more or less persistent until he could not even retain water or milk. Because of the persistent vomiting he had lost approximately 25 pounds in weight. The vomitus was never blood-tinged and he never noted food in it until two or three days previous to admission. He had no tarry stools nor did he note fatty or clay-colored stools.

Physical examination revealed a temperature of 99° F., pulse 132, respirations 24, and blood-pressure 110/70. The scalp, cranium, ears, nose, and



Fig. 46.—Patient on his back.

ST

mouth were essentially negative. The pupils were equal and reacted to light and accommodation. The sclerae were clear and white with no icteric tinge. The expansion of the left side of the chest was definitely decreased. There was dulness up to and including the fifth interspace, with hyperresonance present above. The breath- and voice-sounds were definitely diminished over the affected region. The right side of the chest revealed nothing abnormal. The heart was definitely displaced upward and the apex was visible and palpable in the third interspace in the nipple line. The right heart border was substernal, the left heart border was at the nipple line. There were no murmurs or irregularities. There was very

definite bulging to the left of the upper half of the abdomen. A mass was felt in the epigastrium, smooth, firm, and definitely rounded. It had somewhat of a "doughy feel." There was a suggestive fluid wave in this mass. There was definite dulness in the region of this mass. However, the lower flanks on both sides were normally tympanitic. The mass was slightly respiratory mobile. The liver was palpated approximately 4 cm. below the costal arch in the midclavicular line. It was firm, smooth, and not irregular. There were some visible peristalses in the region of the mass and pulsations in this area presumably transmitted from the aorta. The abdomen was otherwise



Fig. 47.—Patient on his abdomen.

negative. The reflexes were normally present. From these findings a clinical diagnosis of pancreatic or pseudopancreatic cyst was offered.

The vomitus revealed 16 free acid and 31 total. There was no evidence of blood or mucus. The stools were repeatedly negative for occult blood and no further aspirations of the stomach were attempted.

The urine showed no evidence of bile. Albumin and sugar were absent. The microscopic examination was essentially negative.

The blood-count revealed 85 per cent. hemoglobin, 4,700,000 reds and 10,400 whites with a normal differential. The Wassermann test was negative. The blood chemistry showed nothing abnormal. The icterus index was 5.

Fluoroscopic examination of the chest and abdomen revealed that the

left diaphragm was definitely higher than the right. The heart was displaced upward and outward. However, no fluid was seen in either costophrenic angle. The stomach was definitely displaced downward and to the left by a large circular mass and when the patient was in the upright position there was a very definite separation of the stomach in its longitudinal axis at the left, beginning at the cardiac end and extending down to an including the lower third. In the horizontal position and when the patient was on his abdomen this was more marked. However, when he was on his back the stomach remained relatively normal in size and contour except when pressure was made. The duodenal bulb filled out normally (Figs. 46, 47).

With these clinical, laboratory, and roentgenologic findings the patient was transferred to the Surgical Service with a diagnosis of a pancreatic or pseudopancreatic cyst. He was operated on by Dr. Karl Meyer, who found adhesions between the stomach and liver, spleen and liver, and stomach and transverse colon, with a large fluctuant mass between the liver and stomach and definitely connected to the under surface of the liver, displacing the stomach somewhat downward and to the left. Aspiration of this mass revealed a dark brown cloudy fluid. Approximately  $2\frac{1}{2}$  liters of fluid were aspirated from this cyst-like mass. Exploration of the mass revealed that the walls were roughened. It extended backward to the region of the pancreas. Drainage was instituted at the site of aspiration and the abdomen closed in the usual manner.

The wound continued to drain while on the Surgical Service. However, the skin about the incision became "beefy" red. The patient later began to vomit and finally the course went progressively downward. He died twenty-seven days after operation.

Examination of the fluid removed from the mass at operation revealed numerous colon bacilli, but no amebæ or cysts. There were numerous blood- and pus-cells present. Chemical examination revealed that the pancreatic enzymes were normally present, although lipolytic enzyme was definitely diminished.

At necropsy there was found a hemorrhagic pseudocyst of the lesser peritoneal cavity with communication into the pancreatic duct; chronic upper abdominal peritonitis and retroperitoneal pancreatic fat necrosis; perforation of the je-

junum by cyst and emptying of hemorrhagic fluid into intestine; subacute fibrinous peritonitis; partially healed supra-umbilical laparotomy wound with draining abdominal fistula; chronic fibrous mediastinitis; right apical fibrous pleuritis; left diaphragmatic pleurisy; moderate atheromatosis of the aorta and medium-sized blood-vessels; moderate hyperplasia of the peri-aortic lymph-glands; moderate bilateral pulmonary edema, and hyperemia.

Microscopic examination of the tissue excised at the necropsy revealed that this was made up essentially of connective tissue. There was no epithelium lining the cyst wall comparable to that of a pancreatic cyst.

This case is of interest because of many possible clinical diagnoses that were suggested and also because of the origin of this pseudocyst of the pancreas. From the clinical and pathologic findings the patient may have had a localized superficial acute hemorrhagic pancreatitis which later became secondarily infected and finally progressed and produced the mass seen when the patient presented himself to the Medical Service. No other explanation has so far been offered.

#### SUBACUTE BACTERIAL ENDOCARDITIS

**Case III.**—The third and final case is that of a white male, forty-three years of age, who had been on the Medical Service for approximately two and one-half months. His complaint on entrance was weakness, dyspnea and palpitation, and lumbosacral pain. He stated that for four months previous to entrance he had been suffering from the above complaints. These had been unassociated with cough, epistaxis, or hemoptysis. There had been occasionally sharp localized pains over the precordium, some edema of the ankles, disappearing during the course of the night, and a definite increase in pallor for the last six months. He had lost some 12 pounds in weight. There was nothing in the nervous, gastro-intestinal, or genito-urinary history of any importance. He had typhoid-pneumonia in 1908 and some gastro-intestinal disturbance, consisting of epigastric distress and vomiting, about one year previously. At that time he was told he had an ulcer of the duodenum. He had stomatitis six weeks before admission. There was no history of venereal disease. His wife had never been pregnant. His habits were normal. There was no family history of carcinoma, tuberculosis, or diabetes.

The physical examination revealed a well-developed adult white male, not appearing acutely ill, and with normal pulse and respiration and with a blood-pressure of 120/55. The pupils reacted sluggishly to light, but were equal. The conjunctiva was pale. There was no evidence of petechial hemorrhages

in the conjunctivæ or mucous lining of the mouth. The tongue was not smooth or atrophic. There was no generalized lymphadenopathy. The lungs were essentially normal. There was an apical systolic murmur, a short prediastolic murmur at the apex and an aortic systolic murmur. The heart was definitely enlarged to the left. The spleen was palpable but not markedly enlarged. The liver was palpable, smooth, and firm. The extremities showed nothing abnormal. The deep tendon reflexes were exaggerated although the Babinski and associated reflexes were absent. There was no apparent loss of subcutaneous fat.

The urine on entrance contained only a slight amount of albumin, no sugar, casts, or red blood-cells. However, shortly after this examination there were repeated evidences of microscopic blood, although no macroscopic blood was present at any time.

The stomach aspirations showed no free acid, a total of 12 with the Ewald meal, and similar findings on the motor meal with no evidence of retention. The fractional analysis revealed no free acid. The histamin injections brought no secretion of free hydrochloric acid or an increase in the total.

The numerous stool examinations during the whole time that he was in the hospital showed no occult blood.

TABLE 1  
BLOOD EXAMINATIONS

Date.	Hemoglobin, percentage.	Red blood- cells,	Color-index	White blood- cells.	Diff. percentage.	Remarks.
8/15/27		2,240,000	0.9	5,500	Poly. neut. 66 Poly. eosin. 0 Poly. baso. 1 Lymphocytes 33 Monocytes 0	Anisocytosis and poikilocytosis.
8/16/27	60	2,520,000		6,600	Poly. neut. 77 Poly. eosin. 4 Poly. baso. 2 Lymphocytes 14 Monocytes 3	Many macrocytes. Slight poikilocytosis. Polychromatophilia. One myelocyte. No nucleated reds.
8/24/27	47	2,350,000		12,200	Poly. neut. 73 Poly. eosin. 0 Poly. baso. 1 Lymphocytes 20 Monocytes 6	Reticulated cells, 2 per cent. Marked aniso. Poikilocytosis. Polychromatophilia. Achromia.
9/2/27 <sup>1</sup>	30	2,140,000		5,200	Poly. neut. 71 Poly. eosin. 1 Poly. baso. 0 Lymphocytes 5	Polychromatophilia. Anisocytosis. Poikilocytosis. Reticulated cells, 6 per cent.
10/25/27	35	2,350,000		3,800	Poly. neut. 85 Poly. eosin. 0 Poly. baso. 0 Lymphocytes 15 Monocytes 0	

<sup>1</sup> Hemoglobin estimation with the Fleischel-Miescher apparatus.  
Others with the Talqvist scale.

His blood-count was exceedingly interesting. The count on entrance was 60 per cent. hemoglobin, 2,520,000 red cells, 6600 white cells, with a differential of 77 per cent. polymorphonuclear neutrophils; 4 per cent. polynuclear eosinophils; 2 per cent. basophils; 14 per cent. large and small lymphocytes and 3 per cent. transitionals. There were many microblasts, a slight poikilocytosis, and 1 myelocyte. No nucleated reds were seen. His Wassermann test was 1+ positive at one time and negative at another. The urea nitrogen was 34.56; blood-sugar, 1.20; cholesterol, 147 mg. per 100 c.c., and the icterus index, 7.50 (see Table 1).

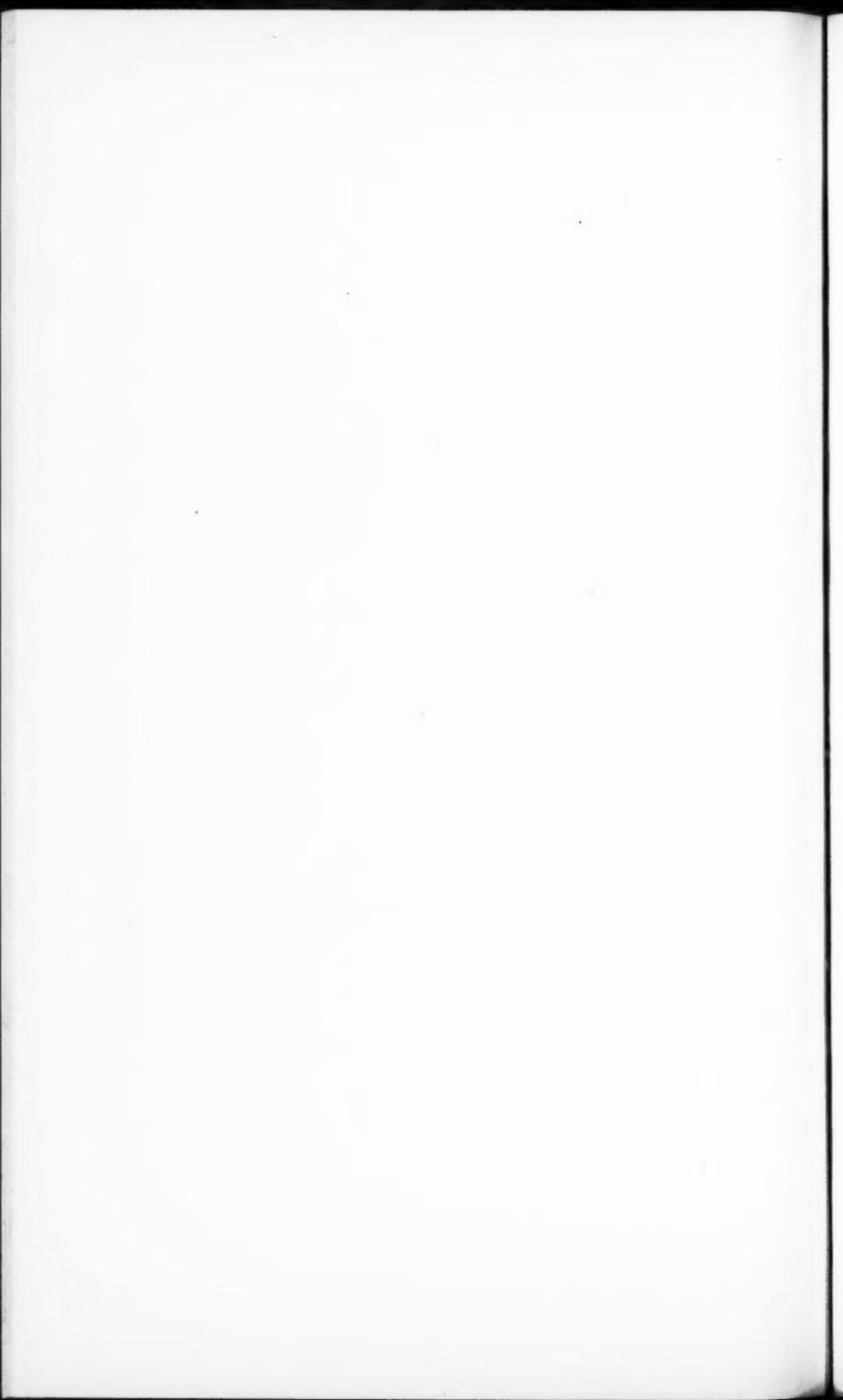
The fluoroscopic examination revealed that the chest was essentially negative. There was nothing of noteworthy importance in the stomach, duodenum, or colon. The plates confirmed the fluoroscopic findings.

At no time during the patient's stay in the hospital was there any evidence of gross hemorrhage. It was noted that the murmur over the aortic region became slightly louder and also that over the mitral region. Three weeks before he died there were petechial hemorrhages evident on the lower conjunctiva and in the mucous membranes of the mouth and at this time the patient became more dyspneic. Repeated blood-cultures previous to this observation were negative. However, at this time *Streptococcus viridans* was isolated. The patient went progressively downward and finally died some two and a half months after entrance to the service.

The necropsy diagnosis was as follows: Acute vegetative exacerbation of a chronic stenosing, calcifying endocarditis with insufficiency of the aortic valve; an old healed endocarditis of the mitral valve; mild myocarditis, and fibrous carditis; hypertrophy of the left ventricle; dilatation of the right cardiac chamber; acute hemorrhagic pericarditis; acute hemorrhagic glomerular nephritis; bilateral hydrothorax and hydroperitoneum; cloudy swelling of the parenchymatous organs; acute splenic tumor; bilateral pulmonary hyperemia and edema; subacute tracheobronchitis; calcified tuberculous focus of the right lower pulmonary lobe; right adhesive pleuritis; luetic scarring of the liver, and fibrous perihepatitis; marked nodular scarring of the spleen; chronic bilateral seminal vesiculitis; pitting edema of both legs.

This case was of unusual interest. It presented many problems of diagnosis and differential diagnosis with this clinical

picture. With absence of fever a diagnosis of primary anemia or aplastic anemia had to be considered. Carcinoma of the gastro-intestinal tract could be definitely ruled out. The rôle of the heart in the causation of the present clinical picture came in for the greatest consideration. From the blood findings it was evident that it was not a primary (pernicious) anemia. The large number of polymorphonuclear leukocytes and the changes in the red cells spoke against an aplastic anemia together with the repeated finding of red blood-cells in the urine. With the severe anemia as presented with its accompanying polynucleosis a diagnosis of a subacute bacterial endocarditis was offered early in the course of the disease and was later confirmed by the clinical and pathologic findings.



CLINIC OF DR. CARROLL LA FLEUR-BIRCH WITH  
PATHOLOGIC REPORT BY DR. R. H. JAFFE

RESEARCH AND EDUCATIONAL HOSPITAL, UNIVERSITY OF ILLINOIS

**CHRONIC HEMOLYTIC ICTERUS IN ADOLESCENCE**

THE patients whom I have to present today are of peculiar interest in that they are all school-boys of approximately the same age. All present a similar clinical picture.

**Case I.**—The first, a white boy (E. D.), aged fifteen. He was born in and has always lived near Chicago. His parents were born in Sweden. He entered the hospital on March 20, 1927 with the following history:

*Present Complaint.*—Tumor in left upper quadrant of the abdomen for three years; attacks of fever with abdominal pain for five years; yellowish pallor during the above-mentioned attacks; frontal headache; sleeplessness, nervousness, and irritability.

*Onset and Course.*—The patient was entirely well until five years ago (1922) when he had a sudden attack of violent abdominal pain accompanied by a high fever and a yellowish discoloration of the skin. So far as the patient can remember, this pain was generalized, and did not radiate to any other part of the body. These symptoms lasted for about three days. From 1922 to the present time (1927) the patient has experienced a number of attacks similar to the one described above. The intervals between attacks were several months. The last attack was in February, 1927, and lasted for four days. We never saw the patient during an attack. During the last five years the patient has grown progressively weaker and paler and there has been a constant enlargement of the abdomen.

*General Symptoms.*—Nervous: Symptoms referable to the nervous system are sleeplessness, nervousness, irritability, and frontal headache. Respiratory symptoms are entirely absent save for dyspnea on exertion. Cardio-vascular: Slight palpitation and progressive pallor are the only symptoms worthy of note in this category. Gastro-intestinal: His appetite is only fair, bowels are a little sluggish and there has been no gain in weight during the last few years. Genito-urinary: Symptoms are absent. Special senses present no abnormalities.

*Past History.*—Patient was one of twins. This was the mother's fifth pregnancy. Patient was a full-term, normal infant, weighing 7 pounds, normally delivered. The other twin weighed 6 pounds at birth and met with an accidental death at the age of three years. Patient sat erect at eight months,

got his first tooth at ten months, walked at fifteen months, talked at eighteen months. He was breast-fed for two years and never was fed from a bottle. He received no orange-juice or cod-liver oil.

*Medical History.*—Patient had whooping-cough at three years, measles at four years, and scarlet fever at five years. *Surgical:* Negative.

*Family History.*—Father met an accidental death. Mother is fifty-seven years old and is fairly well. Three brothers and one sister are living and well. Three children are dead, one died of "summer complaint," one by accident, and one died on the eighth day of life, cause unknown. No tuberculosis or carcinoma in the family and no other member of the family has ever had a condition similar to that of the patient.

*Physical Examination.*—The first impression is that of a poorly developed and undernourished, anemic boy about fifteen years of age, whose abdomen is markedly distended. He is definitely sway-back, to balance the abdominal distention. He assumes the posture of a woman far advanced in pregnancy (Fig. 48). His expression is haggard and weary, and his movements sluggish. He is not acutely ill.

*Regional Examination.*—Hair and scalp substantially negative. Ears: Hearing good, no discharge or mastoid tenderness. Eyes: Sclera shows a definite yellowish discoloration, conjunctiva is pale; pupils react to light and in accommodation; vision is good. Nose: Nares partially obstructed. Mouth: Lips are dry, patient is a mouth breather. Teeth show poor dental and hygienic care; there are several cavities. Tongue shows no abnormalities. Tonsils are hypertrophic and cryptic. Mucous membranes are pale. Neck: Movements are free in all directions. The anterior and posterior cervical glands are enlarged, discrete, and freely movable.

*Chest.*—Is symmetric. The bony prominences are conspicuous. There is depression of the supra- and infra-clavicular fossae and the intercostal spaces. Expansion is decreased, but equal. There is no lag. Litten's sign is present. Lungs: Tactile fremitus is normal. There is normal resonance throughout. Breath-sounds are normal. No adventitious sounds. Heart: Apex-beat is visible in the fifth interspace, 2 cm. to the left of the left midclavicular line. There is a loud systolic murmur at the base of the heart. Heart-tones are audible over the entire chest.

There is a marked widening of the subcostal angle because of the distention of the abdomen.

*Abdomen.*—Markedly distended, with the greatest diameter above the umbilicus. The liver is easily palpated two finger-

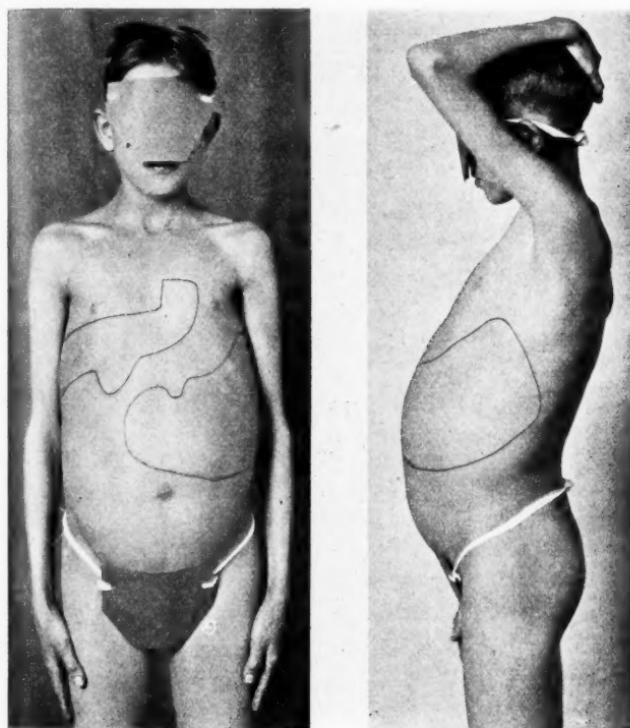


Fig. 48.—Case 1 (E. D.). Showing the splenic borders as obtained by palpation and percussion. Heart and liver dulness are also outlined. Note the prominence of the abdomen and posture in the side view. In the front view observe the general condition and the widening of the subcostal angle.

breadths below the costal border in the right nipple line, it is hard and irregular. Spleen is greatly enlarged and reaches three fingerbreadths to the right of the midline and downward to the level of the umbilicus. An irregularity is easily palpated in its

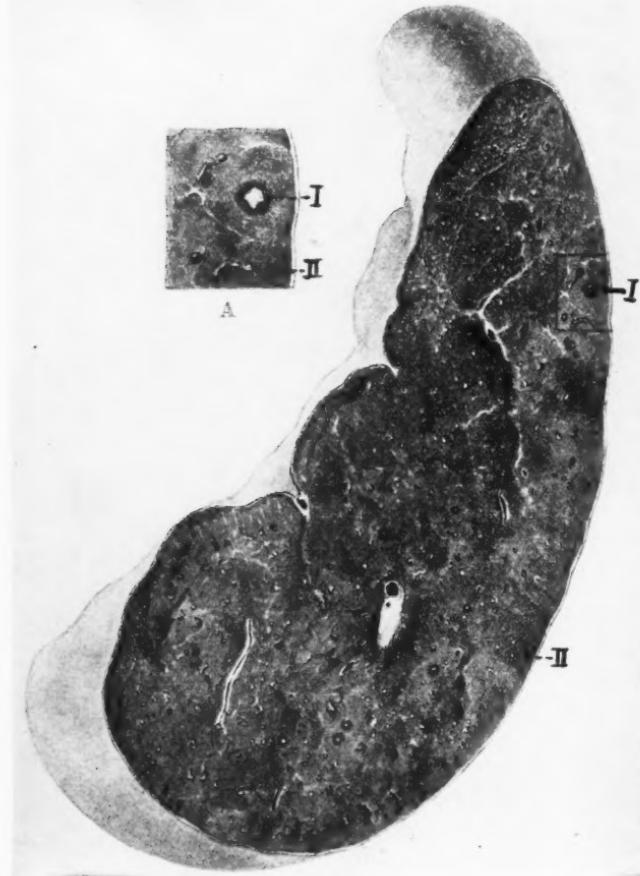


Fig. 49.—Cross-section of spleen from Case I (E. D.). Note the two types of areas. I, The larger ones which are often connected with or located in the trabeculae. These are the seat of fibrotic nodules. II, The smaller spherical nodules representing lymph-follicles, surrounded by hyperemic pulp.

outline. The spleen is hard, slightly movable, and moves with respiration. It is not tender.

*Genitalia* are infantile, the testicles are undescended.

*Extremities* are normal.

*Reflexes* are present and normal, except the upper abdominals, which are absent. No abnormal reflexes are present.

Between March 12th and 26th the patient's respirations were between 20 and 25 per minute, pulse between 60 and 110 per minute, and temperature between 97° and 100° F.

*Laboratory Findings*.—Urine was negative save for an occasional pus-cell. Mosenthal kidney test was normal. Urine



Fig. 50.—Case I (E. D.) with his twin brother at the age of three years. They appear to be quite normal children.

was negative for urobilin (Ehrlich's test). Phenolsulphonephthalein excretion of the kidney was 80 per cent. in the first hour.

Blood: Blood Wassermann negative. Hemoglobin 60 per cent. (by Newcomer). The erythrocytes were 3,750,000, the leukocytes 4100. The differential showed polymorphonuclear neutrophils 54 per cent., small lymphocytes 32 per cent.,

large lymphocytes 12 per cent., transitional cells 2 per cent. The bleeding time and coagulation time were normal. There was no increased fragility of the erythrocytes. Vital staining showed only 2 per cent. reticulated cells.

Liver functional test (dye test) was normal, that is, no dye was recovered after thirty minutes. Icterus index was 22. Van den Bergh: Indirect reaction. Serum bilirubin was 2.6 mg. per cent. Chest was found to be negative on x-ray examination.

After a careful study of the history, physical findings, and results of the laboratory work, we made the diagnosis of "chronic hemolytic icterus in adolescence." We based our diagnosis upon the following findings: 1. The patient's age, fifteen years. 2. The duration of the splenic tumor. It was first observed at the age of twelve years, and had probably been present for a number of years at that time. 3. The anemia. 4. Progressive weakness. 5. Jaundice which was visible in the sclera and which was confirmed by the icterus index of 22. 6. Attacks of acute abdominal pain, accompanied by fever and a deepening of the icterus.

All the conditions causing splenic enlargement were considered, but most of them were easily ruled out. Malaria was not likely, as the patient had always lived near Chicago and as neither fresh blood preparations nor stained blood-smears showed malarial parasites, and patient gave no history of chills. A chronic hidden infection could scarcely produce a spleen of this size. Banti's disease was given careful consideration especially as the liver was large and hard, but because of the condition mentioned above it was thought that congenital hemolytic icterus was the most probable diagnosis.

Splenectomy was performed on March 26, 1927, by Dr. Hedblom. The greater part of the operation was done under local anesthesia. The spleen was skilfully removed and there was very little bleeding. The spleen bed was perfectly dry. The second day after the operation the patient's temperature rose to 103° F. It remained irregular, ranging from normal to 102° F., until April 6th, after which it slowly returned to normal. The leukocyte count increased to 14,000 postoperatively.

The spleen measured 20 x 15 x 7½ cm. and weighed 1315 gm. The normal shape of the spleen was maintained as the enlargement took place in all diameters. At the hilus there was a spherical aberrant spleen 17 mm. in diameter. The consistence was firm and dense, and cut with greater difficulty than normal. There were irregular pittings of the surface. The capsule was gray and diffusely thickened. The cut surface shows recent and old hemorrhages 1 to 2 mm. in diameter. Histologic picture is described at the end of this paper as it was the same for all 3 cases.

**Case II.**—The second patient (P. F.) is an American boy of Italian parents, aged fourteen. He was born and has always lived in Chicago except for six months during his ninth year, which were spent in Italy. He was admitted to the hospital October 31, 1927, with the following history:

**Present Complaint.**—Attack of pain in the upper left quadrant of the abdomen, accompanied by vomiting, frontal headache, progressive weakness, and pallor.

**Onset and Course.**—Patient states that except for an increasing pallor and slight weakness he was entirely well until September, 1927, when he developed a dull, non-radiating pain in the upper left abdomen. The pain became progressively more intense for five or six hours when a physician was called who gave him some medicine to relieve the pain. Soon after the onset of the pain he began to vomit. The vomitus consisted first of partly digested food and later of fluid. It contained neither bile nor blood. The vomiting ceased shortly after the pain disappeared. At this time a large spleen was palpated by the family physician. The patient experienced a similar attack in the middle of October, 1927, and was sent to the hospital following this attack.

**Past History.**—He was a full-term infant, normally delivered. He developed normally and did well at school. In the last few years, however, he has become thinner and paler.

**Medical History.**—He had a mild case of scarlet fever when very young. "Blood-poisoning" following an injury to his knee at the age of ten years. Pneumonia several years ago.

**Surgical History.**—Tonsillectomy at the age of six years.

**Family History.**—Parents living and well. Three brothers and sisters are well. No children dead. No tuberculosis or carcinoma in the family and no member of the family has had a condition similar to that of the patient.

**Physical examination** reveals a pale, fairly well-developed, undernourished, Italian boy whose abdomen is slightly distended.

*Regional Examination.*—Scalp and hair normal. Ears: Hearing good, no abnormalities. Nose: Partial obstruction, no discharge. Eyes: Scleræ show slight yellow tinge, conjunctiva is pale, pupils react to light, and in accommodation; extra-ocular movements good, vision unimpaired. Mouth: Lips are dry, patient is a mouth breather. Teeth in good condition. Tonsils out. Neck: Movements free, anterior, and posterior cervical glands are discrete and movable.



Fig. 51.—Case II (P. F.). Observe the contour of the chest, the prominence of the bony parts, the widening of the subcostal angle, and the general condition of the boy. The approximate size and location of the spleen are indicated.

*Chest.*—Symmetric, expansion fair, and equal. Supra- and infra-clavicular fossæ and intercostal spaces are depressed. The subcostal angle is widened. No abnormal lung findings. Cardiac borders are within normal limits. There is a loud, harsh, systolic murmur at the base of the heart, and a loud venous hum at the inner end of the right clavicle.

*Abdomen* is slightly distended with the greatest prominence

above the umbilicus. Spleen is four fingerbreadths below the costal border. It is firm, smooth, not tender, and moves with respiration. The notch can be palpated. The liver is two fingerbreadths below the costal border in the nipple line. It is firm and not tender. Axillary and inguinal glands are palpable, discrete, and fully movable.

*Reflexes* are all present and normal except the upper abdominals which are absent. No abnormal reflexes are present.

*Extremities* are normal.

*Laboratory Findings*.—Urine negative save for a faint trace of albumin. Negative for urobilin. Phenolsulphonephthalein kidney test showed 55 per cent. excretion in first hour and 20 per cent. in second hour.

Blood Wassermann and Kahn tests negative.

Blood chemistry 105 mg. of sugar in 100 c.c. blood; 45 mg. of non-protein nitrogen in 100 c.c. blood. Hemoglobin 58 per cent., erythrocytes 3,624,000; leukocytes 3000. Differential showed 50 per cent. polymorphonuclear neutrophils, 39 per cent. lymphocytes, 3 per cent. large mononuclears, 7 per cent. eosinophils, 1 per cent. basophils.

Fragility test: Hemolysis began at 0.44 per cent. and was complete at 0.32 per cent. Bleeding and coagulation times normal. No increase in reticulated cells. Icterus index 22. Van den Bergh: Indirect reaction. Serum bilirubin 5.2 mg. per cent.  $x$ -Ray of chest was negative.

In this case the same diagnosis was made as in that preceding and for the same reasons. Because of the patient's visit to Italy a more thorough search was made for malarial parasites, but none were found.

The spleen was removed on November 15, 1927. Because of many anomalous vessels, near the hilus of the spleen, which were very friable, there was much bleeding. Immediately following the operation there was a transfusion of 500 c.c. of citrated blood. For ten days the patient had an irregular fever reaching 103° F. On November 25th, a puncture was made at the level of the ninth rib in midaxillary line and 135 c.c. of purulent greenish fluid were removed, the patient had a subphrenic abscess. On December

15th a portion of the eighth rib was resected to promote better drainage. From this time the patient improved steadily and has returned to school.

The spleen measured  $20 \times 12\frac{1}{2} \times 7\frac{1}{4}$  cm. and weighed 722 gm. The cut surface showed recent and old hemorrhages in the trabeculae with irregular iron deposits around the older hemorrhages.

**Case III.**—The third patient (C. W.) is an American boy, aged fourteen, and, save for pallor, looks like a normal boy. He entered the hospital November 14, 1927, giving the following history:

*Present Complaint.*—Weakness which has been progressive for three years; shortness of breath on exertion and sharp abdominal pain on exertion, especially when running.

*Onset and Course.*—Patient states that he was entirely well until three years ago. He says that he could swim a longer distance than most boys of his age and that he frequently ran three miles to school. During the summer of 1926, he noticed that he could not swim so far as he could the year before. He became short of breath and was forced to leave the water. He also noticed that he could not run so far. In the summer of 1927, he could not swim at all; immediately on entering the water he became so short of breath that he could not remain. At about this time he first noticed the sharp abdominal pain on exertion.

In August, 1927 he was operated on for a right inguinal hernia. On leaving the hospital he became progressively weaker and his activities became more and more limited.

*Past History.*—Patient was born and has always lived near Chicago. He was a normal, full-term baby and developed normally.

*Medical History.*—Patient has had measles, chickenpox, whooping-cough, scarlet fever, and pneumonia.

*Surgical History.*—Herniotomy in August, 1927.

*Family History.*—Mother in hospital for insane; father is a hard drinker and is now in the county jail. There are four other children in the family, all of whom are well.

*Physical Examination.*—Reveals a rather small, pale, but well-developed and nourished boy about fourteen years of age.

*Regional Examination.*—Scalp, hair, nose, and ears normal. Eyes show slight yellowishness of sclera and pale conjunctiva. Lips, tongue, and teeth normal. Tonsils enlarged and cryptic. Neck movements free, anterior, and posterior cervical glands palpable.

*Chest* is symmetric, the expansion is fair and equal. Lungs: Tactile, fremitus is normal, resonance is good throughout, no

adventitious sounds. Heart: Borders within normal limits. Heart-tones normal. No murmurs or thrills.

*Abdomen*.—Well rounded, the upper half is more prominent than the lower half. There is a small herniotomy scar in the lower right quadrant. The liver is palpable four fingerbreadths below the costal border in the nipple line, it is hard and slightly irregular. The spleen is palpable two fingerbreadths below the costal border. It is slightly movable and moves on respiration. Axillary glands are not palpable. Inguinal glands are palpable.

Reflexes are normal and present.

*Laboratory Findings*.—Urine negative.

Blood and spinal Wassermann: Negative.

Blood chemistry showed sugar 92 mg. per 100 c.c. of blood and non-protein nitrogen 33 mg. per 100 c.c. of blood. Hemoglobin 64, erythrocytes 3,950,000, leukocytes 6150. Differential: Polymorphonuclear neutrophils 42, small lymphocytes 30, large lymphocytes 21, and large mononuclears 3.

Bleeding and coagulation time normal. Fragility test: Hemolysis began at 0.46 and was complete at 0.34.

Liver function test (dye test, bromsulphthalein) was negative; that is, no dye was obtained after thirty minutes. Icterus index 7.5. Van den Bergh: Direct—negative. Serum bilirubin 1.2 mg. per cent. Vital staining showed no increase in the number of reticulated cells.

On this patient we made the same diagnosis of congenital hemolytic icterus. It is a similar case, although earlier than the other two.

Splenectomy was done by Dr. Hedblom on January 10, 1928. There was very little bleeding, the spleen bed was perfectly dry. Following the operation he had a moderate elevation of temperature for a number of days. The patient is still in the hospital. He is up and about the ward and is improving steadily. The spleen measured  $11\frac{1}{2} \times 3 \times 9\frac{1}{2}$  cm., and weighed 278 gm. The normal shape was maintained.

The following was dictated by Dr. Jaffe and gives the histologic picture of all three spleens.

The histologic picture of all the three spleens showed es-

sentially marked engorgement of the pulp especially of the cords. The sinuses were rather narrow with slightly hypertrophic endothelium. There were areas of granulopoesis with many eosinophilic leukocytes and myelocytes. The malpighian bodies were large and showed a distinct differentiation into three zones, namely, a center of lymphoblasts, a medial zone of lymphocytes, and an outer zone of large lymphoid cells. There were numerous recent and old hemorrhages into the trabeculae with deposits of iron pigment and scleroses of the connective tissue. In these areas structures were found suggesting mycelium and heads of aspergillus. These structures were often seen engulfed by giant-cells. They were identical with those described by Nanta and Pinoy, also by Weil and Askanazy in cases of so-called mycotic splenomegaly.

Histologic report of biopsy section of liver from Case III (C. W.). No liver sections were obtained from the other 2 cases.

Near the lower margin there is a very marked increase of the periportal connective tissue. It surrounds irregular groups of liver-cells without a central vein. The connective tissue shows numerous round or oval, pale-stained nuclei which have no chromatin net and one or two small nucleoli. There are many lymphocytes and single plasma-cells. In some places polymorphonuclear leukocytes are predominating. They also invade adjacent liver-cell cords. There is a proliferation of small bile-ducts. The liver-cells which form the islands have a loose cytoplasm which often is filled with pigment deposits. There are numerous small, dark-brown granules, and casts formed of a thick, waxy dark-green material. Both types of the pigment are found together in the same cell. Single liver-cells with a very loose cytoplasm show no nuclei, others which are free from pigment have two or three distinct nuclei. There are many mitotic figures. In some places the liver-cells arrange themselves about a central lumen which is empty. The intercellular bile-ducts often are filled with a thick dark-green cast. The Kupffer cells contain dark-brown pigment; a few of them also contain erythrocytes. The capillaries are very narrow. Some distance from the lower margin the changes are much less pronounced.

There is just a slight thickening of the periportal tissue with small round cells loosely arranged. The liver-cells have the appearance of large glycogen-filled elements with very distinct membranes. Occasionally a swollen Kupffer cell is found containing an engulfed red blood-corpuscle.

*Anatomic Diagnosis.*—Early cirrhosis of the liver.

Congenital hemolytic icterus, although not rare, is sufficiently uncommon to make these 3 cases of peculiar interest, especially as they were admitted to the hospital in rather close succession. They are also interesting in that they present three stages in the development of this disease. The first case was the most severe and was probably of longer duration than the other two. It would be of real value to have known the condition of his twin brother at the age of three years. Figure 50 shows the patient with his twin brother just after their third birthday. We have other pictures of the patient taken at seven months, four years, and six years, none of which show any gross abnormalities. It is unfortunate that we did not see the patient during one of his attacks of abdominal pain and fever for it is during these times that there is the greatest blood destruction, when the fragility of the erythrocytes is greatly increased and the percentage of reticulated cells is high. The icterus also deepens during these attacks, this phenomenon having been noticed by the patient's family. The two most outstanding symptoms are persistent icterus, which varies in intensity, and acute attacks of abdominal pain. In this case the spleen was unusually large, weighing 1315 gm. The weight of a normal spleen of a boy of his age is 145 gm. The spleen was therefore nine times the normal weight. The great size of this spleen would suggest a leukemia. Examination of the blood, however, quickly ruled this out.

This disease suffers under the burden of many names a few of which are congenital hemolytic icterus, chronic hemolytic jaundice, familial hemolytic jaundice, constitutional jaundice, and acholuric jaundice. Perhaps the most applicable name is chronic hemolytic jaundice as the disease may be congenital, acquired, or familial. In the congenital type, it may affect only a single member of the family. It is practically never seen in

older persons and is far more common in males than females. The anemia is usually moderate, but it may become of high grade in severe cases, sometimes approaching the pernicious type.

Our second patient (P. F.) had had symptoms for only two months when he entered the hospital. He had experienced only two attacks of abdominal pain. It is impossible to state how long his spleen had been enlarged. His family doctor does not believe that it has been enlarged since birth. His blood-picture was almost identical with that of patient Number I; so if we assume that his disease was of shorter duration, it was a more severe type in that it produced the same degree of anemia in a much shorter period of time. The spleen in this case was considerably smaller, weighing 722 gm., or about five times the normal size. It has been stated that the greatest increase in the size of the spleen occurs during the crisis. These 2 cases would bear out that observation as the first boy had had many crises and a massive spleen, while the second boy had had but two crises and a much smaller spleen. After splenectomy, P. F. developed an abscess. The aspirated pus was carefully examined for the mycelia-like structures described by Dr. Jaffe, but none were found. Culture of this pus revealed only staphylococci.

The third patient (C. W.) presented an early stage of this disease. He had had no crises and his anemia was not so severe. The fragility of the red blood-cells was tested a number of times and only once was there an increase in fragility, at this time hemolysis occurred at 0.5. In this instance the spleen weighed only 278 gm., not quite twice the normal weight.

These three are quite typical cases, and show clearly the progress of the disease by the 3 stages represented. Splenectomy was first performed for hemolytic icterus by Dr. Banti in 1903. Since that time many reports have confirmed the curative results of splenectomy in this disease. At the present time our 3 patients are all doing well.

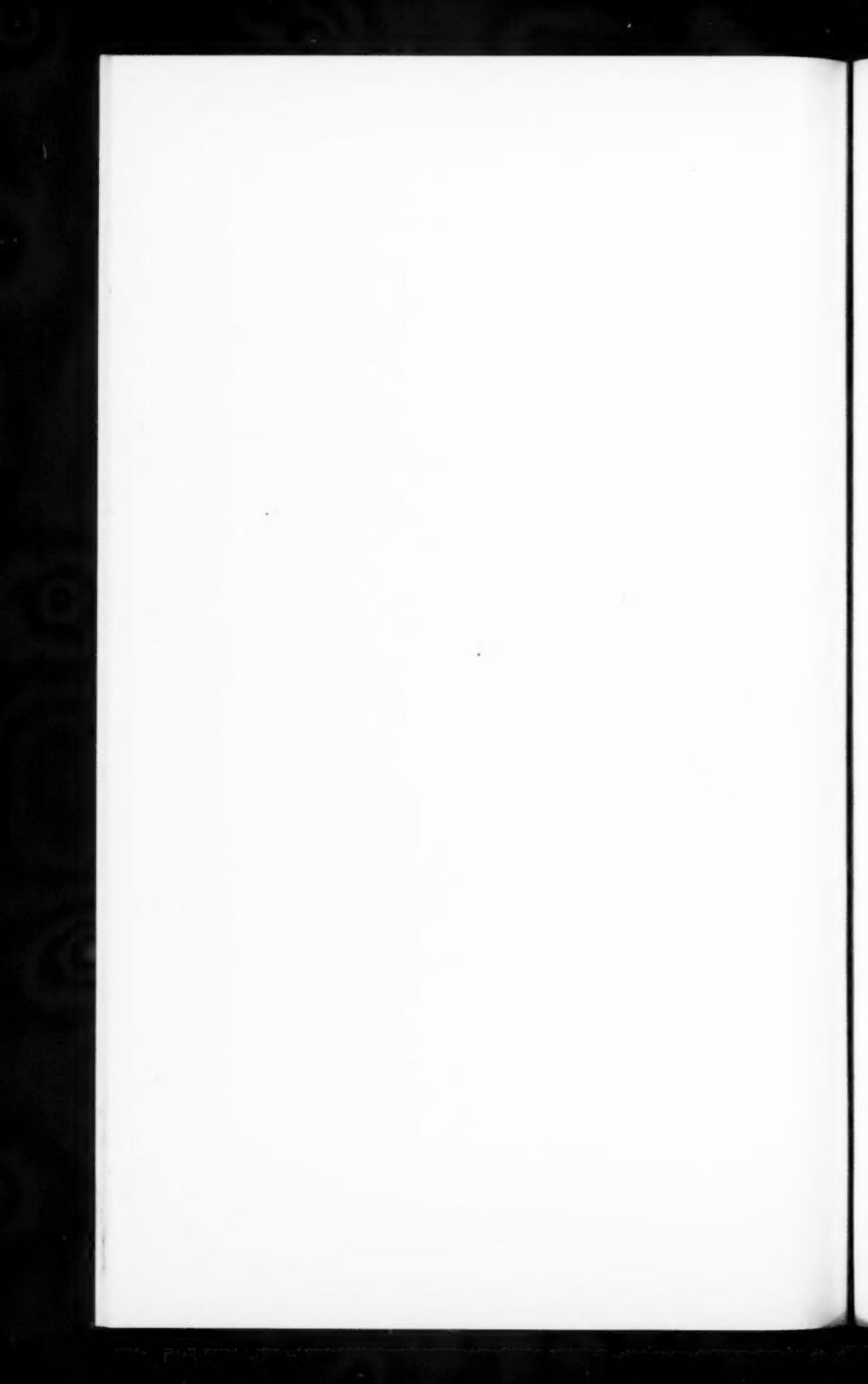
The exact significance of the presence of the fungus-like structures found in these spleens by Dr. Jaffe cannot be stated at this time, as he has not completed his study.

Since this article went to the publisher all of our patients have been heard from.

**Case I** (E. D.), one year after splenectomy, has gained 17 pounds in weight (former weight 78 pounds, present weight 95 pounds). Hemoglobin is 79 per cent., an increase of 19 per cent., he has 4,720,000 red blood-cells, an increase of one million. We requested this patient to return to the hospital to have his testicles brought down. Upon examination we were surprised to find both testicles in the scrotum. Apparently after splenectomy the interabdominal pressure was decreased, which allowed the canals to open and the testicles to descend. He looked like a new boy, much more alert and interested in everything.

**Case II** (P. F.), five months after splenectomy, has gained 13 pounds in weight (former weight 116 pounds, present weight 129 pounds). His hemoglobin is 78 per cent., an increase of 20 per cent. Red blood-cell count is 4,110,000, an increase of 500,000.

**Case III** (C. W.) we have not seen, but have received three letters from him, and he states that he is feeling well and gaining in weight.



## CLINIC OF DR. ELLIS KIRK KERR

### COOK COUNTY HOSPITAL

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#### A CASE OF ATROPHIC CIRRHOsis OF THE LIVER

THIS patient, who is thirty-eight years old and a laborer by occupation, entered the Cook County Hospital November 11, 1927. He gave the following history on admission:

About August 15th he noticed he was jaundiced and consulted his family physician, who told him he had an enlarged liver. The patient states that his stools were clay-colored. He continued at his work for a week when he became so very weak that he could hardly move his hands. He had no other symptoms than this excessive weakness. About the first of November, he found that his abdomen was swollen and about 1 quart of fluid was removed by paracentesis. He has been tapped several times since on account of recurring fluid. He gives no cause for the occurrence of the jaundice. He has used alcohol at frequent intervals for ten years, drinking a pint, one to three times a month, but had been on no spree prior to the onset of the jaundice nor had he had any sort of gastro-intestinal upset. His previous history is negative for any causative factor and the family history is negative. He denies having had lues.

On examination we note that he is well nourished and does not look particularly ill. He has a peculiar bronze color due to a moderate degree of jaundice in a patient with a natural dark complexion. His sclerae are yellowish. Pulse and temperature are normal. We find no abnormalities except the color on examination of his head and neck. His chest is normal in contour, and heart and lungs are negative. Examination of the abdomen shows the presence of free fluid in the abdomen, but neither now nor after paracentesis can any enlargement of the liver and spleen be made out, nor are there any other abnormal findings in the abdomen. Examination of the extremities shows a moderate edema, but no other findings. The reflexes are normal. Blood-pressure is 118 systolic and 82 diastolic.

Examination of the urine shows no albumin nor casts, but there is some bile present. The stools have always contained bile, and between November 22d and December 7th, blood was found, but none has been found since on repeated examinations. Wassermann test was negative. Blood-urea nitrogen and sugar were normal. Blood examination showed hemoglobin 60 per cent., red corpuscles 3,500,000, leukocytes 3900. Differential counting showed 48 of polymorphonuclear leukocytes, 44 per cent. lymphocytes, and 7 per cent. mononuclears and transitionals. There were no abnormal constituents nor any variations in size or shape of the red corpuscles. *x*-Ray examination of the gastro-intestinal tract is negative. Examination of the fluid removed by paracentesis showed some bile staining, but the fluid has all the characteristics of a transudate.

We have then a patient presenting himself with a history of what would seem to have been a catarrhal jaundice in August. Examination at present shows a moderate degree of jaundice and free fluid in the abdomen. He has a secondary anemia of moderate degree, but no nephritis, nor cardiovascular disease and no local findings of abdominal disease to account for the ascites. In the absence of any general and local cause for the ascites we are justified in making a diagnosis of cirrhosis of the liver. I am going to step further and venture the opinion that the period of weakness complained of during the early part of his attack of catarrhal jaundice, was in reality due to an acute degeneration of possibly, we may say, atrophy of the liver. He survived this acute degeneration, but enough destruction of the liver resulted to produce portal obstruction—in other words, what we know as atrophic cirrhosis.

It is unfortunate that certain fixed ideas dominate the general impression of atrophic cirrhosis of the liver. It is supposed to be always due to alcohol. The idea is general that the liver is small except that it may be enlarged early and will contract later and there is considerable confusion regarding a so-called hypertrophic cirrhosis characterized by enlarged liver and spleen, together with jaundice. With a clearer idea of the pathogenesis of the disease, one gets a broader outlook and a condition which is

but relatively uninteresting comes to be of great importance. It is the occasional case such as the present one that makes cirrhosis important to the clinician. In order to bring this out, a review of the salient points in the pathology of cirrhosis is in order:

1. Atrophic cirrhosis is due to a primary degenerative change in the liver.

2. Following this degeneration, there are two definite and distinct changes in the liver. There is, first, a replacement of the destroyed tissue by fibrous tissue with a resulting scarring of the liver giving rise, in marked cases, to the granular, hobnailed surface so characteristic of the process. There is, second, and more important, a varying degree of regeneration of liver-cells. In the milder grades this regeneration proceeds from the liver-cells and if the distinction of the latter is not marked, there may be a complete *restitutio ad integrum*. In the severe cases, however, this regeneration takes place probably from the bile capillaries and gives rise to small ovoid masses of cells which may be easily noted on the cut surface of the liver. They are usually bile-stained and stand out as irregularly oval, yellowish areas varying in size from a small pea to a cherry. In the latter case they are spoken of as adenomata. Rarely do the regenerated cells revert to the fetal type and give rise to the primary carcinoma of the liver. This type occurring in cirrhosis is perhaps the most common type of primary cancer of the liver. These regenerated liver-cells are bile-stained because they have not the intimate relation to the bile vessel that is characteristic of normal liver-cells. They are functionally active, but lack the means of getting rid of the bile they secreted. They are functionally inefficient too because they lack the intimate relation to the vascular system possessed by normal liver-cells. Hence, even markedly regenerated livers are functionally inefficient. As a rule the degeneration and regeneration do not occur once only, but the process is repeated time and again, leading to most bizarre pictures. This is the reason that so many types of cirrhosis have been described. The size of the liver depends on the amount of regeneration. It may range from half the size of the normal liver to three to four times its size. Such enlarged livers do not contract, but remain

enlarged throughout the course of the disease. The liver is larger than normal in over half the cases of atrophic cirrhosis. You will find this statement in Osler's Practice of Medicine.

3. Portal obstruction is the distinguishing clinical feature of atrophic cirrhosis and whenever it occurs this diagnosis must be made in spite of enlargement of the liver and spleen, jaundice, etc. The so-called Hanot's cirrhosis is extremely rare and it should only be diagnosed when an enlarged liver and spleen are found together with recurrent attacks of jaundice, fever and leukocytosis, over a period of many years. There is no ascites or, at any rate, none till the terminal stages. These cases are probably due to chronic cholangitis with recurrent acute exacerbations. There is a biliary cirrhosis described with large tender liver and jaundice. As a rule this is a local process limited to the region of the gall-bladder.

4. Jaundice frequently occurs in atrophic cirrhosis due to an ordinary common duct catarrh or possibly to a subacute or chronic cholangitis. In the latter case, it might be proper to speak of a mixed cirrhosis. It is better, however, to consider every patient with obstruction as an atrophic cirrhosis with a complicating cholangitis. The important matter is to hold the underlying pathology in mind. In the one case there is degeneration of liver-cells, in the other a chronic cholangitis.

The most important fact to remember about atrophic cirrhosis is that it is always the result of a degenerative process in the liver and that such processes are common and are due to a variety of causes. In the patient shown today the process probably occurred in the course of an ordinary catarrhal jaundice though it is, of course, impossible to say that the liver was entirely normal previously. It may have been damaged to a certain extent by alcohol. Such an acute degeneration occurs not infrequently in the course of a catarrhal jaundice. Osler speaks of it as an *icterus gravis* and mentions the possibility of its occurrence. Once in a while the patient dies, but at times cirrhosis results and the relation to the preceding *icterus* is overlooked. It will not be overlooked if the relationship between the two conditions is borne in mind. There is not infrequently an

acute cholangitis with fever, enlarged tender liver, etc., which is akin on the one hand to catarrhal jaundice and may approach what is called acute infectious icterus on the other. In this kind, Osler calls attention to the occurrence of *icterus gravis*. In patients with atrophic cirrhosis an *icterus gravis* may develop with an early exitus. We had such a case recently on one service where at autopsy an acute degenerative process was found, together with the older changes of cirrhosis.

An acute atrophy may occur in the course of any of the acute infections. It is a rare complication of typhoid fever, but has been described. It occurs in the so-called secondary stage of syphilis, and in that case will show no characteristic luetic lesions, but is a manifestation of the acute general infection. It will be hard to say whether it is due to the lues or to the treatment in those patients treated by the arsphenamin preparations, for the same process occurs with this group of drugs. It occurs in particular as a result of local inflammatory processes in the abdomen, such as appendicitis, local abscesses, general peritonitis, etc. I remember a patient of twenty-one years who was operated on for bilateral inguinal hernia. One side became infected, requiring about three months to heal, by which time there was ascites present. This was several years ago and we thought that the cirrhosis was possibly due to a combination of infection and chloroform with which anesthesia was begun.

Active liver degenerations occur as a result of poisoning by various drugs, particularly the heavy metals. Arsenic has been mentioned. Mercury may be a cause. Phosphorous poisoning shows as a characteristic change, acute necrosis of the liver and in those cases surviving a few weeks beginning fibrosis has been noted. Chloroform has been mentioned. There is a group of the toxemias of pregnancy, where the liver is particularly affected. In this connection a patient recently seen at the West Suburban Hospital with Drs. Post and Van der Slice may be mentioned.

A child just under six years had the signs of a rather rapidly developing ascites. After paracentesis, the liver was felt and was very firm. The child had always been well and there was no

evident reason for the fluid. It was found, however, that at the child's birth the mother had had eclampsia, described by Dr. Carey Culbertson as being of the hepatic type. As it is well known that the fetus has the same lesions, a diagnosis of atrophic cirrhosis was made. This diagnosis was confirmed by an exploratory laparotomy when the liver was found to have the typical surface described by Laennec. The child had an acute atrophy at birth, but survived, thrived on the simple diet of the first few years, but was unable to handle the increased demands on the liver with increasing age. Such a case would be unsolvable with the general idea of alcohol being the only cause of cirrhosis.

The association of atrophic cirrhosis and splenic enlargement appears to be very close. Splenic anemia frequently terminates in atrophic cirrhosis; for example, it would appear that toxins develop in such an enlarged spleen which produce degeneration in the liver. Incidentally even in the ordinary type of cirrhosis, that due to alcohol, this may be the sequence since alcohol by itself has not been proved to be particularly toxic to the liver and the spleen is usually enlarged out of proportion to the portal stasis.

You see now how intensely interesting the subject of atrophic cirrhosis is when you get away from the stereotyped view of its always being due to alcohol. The occasional case which you will meet acquires a new interest and such instances will be more frequent if you will be on the lookout for them. Instead of being the exceedingly rare condition that is usually emphasized, acute degenerative processes in the liver occur not seldom. They are clinically important because they may be a cause of death or lead to invariable scarring of the liver. They are to be borne in mind when giving arsenicals and in the handling of such conditions as catarrhal jaundice. In connection with the latter group, a therapeutic idea grows out of the experiences of experimental medicine. When the early attempts were made to make an Eck fistula in dogs, the animals all died. When, however, after operation they were kept on a bread and milk diet, many of them lived. One of the most important functions of the liver is to detoxicate materials absorbed from the gastro-intestinal tract

so when the function of the liver is disturbed the diet should be simple with a minimum of amido bodies requiring removal.

To return to the patient, he is being tapped as needed and is on a diet that throws only a limited strain on the liver. He is being kept in the hospital to protect his liver as much as possible. There is a chance that by regeneration of new tissue and by a quieting down of the cholangitis responsible for his jaundice, he may return to a fairly normal condition and again become a wage earner. His age makes this more likely than in the ordinary patient, and the fact that he has suffered only one rather than a series of liver destruction. It is surely worth while giving him the benefit of a chance at repair.

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## CLINIC OF DR. SAMUEL J. TAUB

COOK COUNTY HOSPITAL

### PRIMARY BRONCHOGENIC CARCINOMA OF THE LUNG

INTRATHORACIC neoplasms, especially carcinoma of the bronchi, lungs, pleura, and glands, are often mistaken for tuberculous disease of the lungs. The onset is insidious with cough and mild fever, the curve in some cases simulating that seen in typical cases of incipient tuberculosis. When hemoptysis of various degrees and loss in weight occur, it is clear that tuberculosis is the disease thought of at once since it is the more common disease. Malignant disease of the lung is likely to pursue a very slow course. The differentiation is made by the symptomatology, the physical signs, and the x-ray, though I consider the x-ray findings less reliable in early cases than interpretation of signs elicited by a careful physical examination. In carcinoma of the lung, less than 2 per cent. of all cases are primary. The onset is usually insidious with cough, which at first is non-productive, later being followed by mucopurulent and bloody sputum, dyspnea increasing in severity then follows, and intrathoracic discomfort and pressure is noticed, usually after the tumor has reached a fairly large size. Pain is a constant symptom and is usually present in the chest or shoulder and even tenderness of the skin of the affected side may be complained of. Compression of the esophagus and the recurrent laryngeal nerve may cause dysphagia and hoarseness. Pressure on the ciliospinal branch of the sympathetic leads to pupillary inequality. In the later stages the general symptoms of malignancy are present, such as cachexia, pallor, weakness, loss of weight, loss of appetite, etc. Extension of the growth to the vertebral column and cord may by encroachment, or invasion, produce a pressure myelitis.

At the beginning of the disease, when the new growth is still small, the physical signs may show no alterations in the resonance and breath-sounds, and the constitutional symptoms may confuse one with tuberculosis. However, in nearly all cases, even those showing a febrile temperature, the pulse-rate is normal, which is rare in tuberculosis. With the growth of the tumor, local signs may be elicited by physical examination. Percussion over the site of the tumor elicits a flat note, while above and below this area of flatness a resonant note is present. If the tumor is of some dimensions, auscultation reveals feeble or absent breath-sounds over the circumscribed area of flatness. These two physical findings are of the utmost significance and strongly suggestive of tumor, especially with the absence of râles. The reasons for the absence of breath-sounds are: The tumor arises from the wall of a large bronchus and as it grows it compresses the air-tube and the result is atelectasis of the lobe supplied by this bronchus, giving the signs of flatness, absent breath-sounds, and no râles. With the growth of the tumor, the area of flatness increases, the veins on the chest become more prominent and at times metastases occur in the glands above the clavicles. An important finding is the displacement of the trachea and heart toward the unaffected side. In the later stages of cancer when the tumor becomes gangrenous and fetid sputum is expectorated, the mediastinum may be drawn toward the affected side. In over 50 per cent. of cases of malignant tumor of the lung or pleura, effusion occurs. The effusion may be serous, serosanguineous, purulent, or sanguineous. At times fragments of the growth or characteristic cells are found in the centrifugal specimen of the aspirated fluid, but this is rare except where the growth involves the pleura. One characteristic of effusions in cases of cancer of the lung is that they fill the entire pleural cavity.

The pathology of primary pulmonary carcinoma is most interesting. The rarity of the disease is somewhat difficult to comprehend since in the lungs and bronchi there is such a preponderance of tissue of epithelial structure. There are three types of epithelial cells: The columnar epithelium lining the bronchi and larger bronchioles, the flattened or cuboidal epithe-

lum lining the alveolar spaces, and the glandular epithelium found in the mucous glands. The epithelium lining the pleura may also be mentioned as a possible source of primary carcinoma of the lung, but will not be discussed in this paper. In most instances the carcinomas originate from the columnar cells lining the larger bronchi. The favorite site of origin is said to be in the second or third division of the main bronchus, but any portion may be first involved. The primary nodule enlarges, invades the bronchial wall, and extends out into the smaller bronchi and into the parenchyma of the lung. The bronchial lymph-glands are usually involved, but numerous cases are on record in which they showed no evidence of metastases, although both lungs were affected. There seem to be no definite data in regard to the frequency and the favorite sites of metastases outside the thoracic cavity, unless it be the axillary and cervical lymph-glands. The brain may be mentioned as a probable, and at times the only, site of metastases in primary lung carcinoma. Grossly there are three types of primary carcinoma of the lungs and bronchi. The infiltrative, the miliary, and the mixed types. The infiltrative type is the most common. The condition starts in one of the larger bronchi as a nodular tumor of varying size. It penetrates the wall and invades the lung along the bronchi and bronchioles, the alveolar walls and air-spaces by way of the blood-vessels and lymphatics, by direct extension, by gravity, and by aspiration. A single lobe may be affected, usually the lower, or there may be massive areas involving parts of both lungs. In the miliary type the nodules are very numerous, two to ten or more times the size of macroscopic miliary tubercles, they have a more or less symmetric and diffuse distribution throughout both lungs, and are found far out in the periphery as well as the central parts. The pleura may be involved with carcinoma or with simple chronic inflammatory changes. The tumors are homogeneous in form, grayish-white in color, invasive in character, not sharply demarcated from the surrounding lung tissue, and firm on section. Several of them may coalesce to form larger, irregular nodules. Degeneration and central necrosis sometimes occur, leading to caseation and cavity formation. The mild type presents both

the infiltrative and the miliary forms in the same case. In addition to a large and hard, wedge-shaped or nodular area in one or more lobes, the remainder of the lung is diffusely studded with miliary nodules varying in size and number, depending on the duration of the disease.

In the Roentgen examination three types of the disease are recognized: namely, the infiltrative, the miliary, and the mixed which correspond to the gross pathologic groupings. A striking feature in all types and one of considerable diagnostic importance is the absence of practically any increase in mediastinal density. The presence of extensive pleural involvement or pleural effusion marks the roentgenographic picture.

In the stereoscopic study of the infiltrative type the roentgenogram shows one or more areas of increased density along the roots of the larger bronchi. The shadows are homogeneous or partially mottled. The borders are infiltrated and not sharply demarcated. The areas of density are wedge-shaped, with the apex pointing toward the hilus, and there may be either unilateral or bilateral involvement. The degree of density is marked, but varies with the extent and duration of the disease. The most frequent site of this type of lesion is in one of the lower lobes. In the miliary type, there are innumerable, regular, irregular, or conglomerate small areas of increased density, extending throughout all the lobes. Their borders are clearly defined, and not sharply circumscribed from the surrounding parenchyma of the lung because of the marked infiltrating character of the neoplasm. The process is diffuse throughout both lungs and the areas of density are distributed as uniformly near the hilus as in the periphery of the lung. The shadows show no tendency to be arranged in groups or clusters. There are usually no true cavities. The mixed type includes a combination of the infiltrative and the miliary types. In this type are found poorly circumscribed, homogeneous, or slightly mottled areas of increased density, similar to those found in the miliary types, diffusely studding the entire remaining portions of both lungs.

The case report with autopsy findings of a typical infiltrative type of primary homogenic carcinoma of the lungs follows:

M. Z., aged fifty-four, single, white, Polish nationality, entered the Cook County Hospital, June 10, 1927, with the following complaint:

Pain in the chest, pain in the abdomen, cough, expectoration. An examining-room diagnosis of far-advanced pulmonary tuberculosis was made. Very little history was obtainable because the patient spoke little English. Cough and expectoration have been present on and off for the past two years. Since the last seven months the cough has been constant and seems to be getting worse. Pain in the chest which he is unable to describe; he points vaguely over his abdomen to indicate discomfort of some kind there.

Physical examination reveals a poorly nourished white male, about fifty years of age, who does not appear acutely ill. Temperature 98° F., pulse 96, respirations 26. Pupils are equal, react to light and accommodation. Nose, throat, and mouth are negative.

Chest: Flatness over the right lower lobe, absent breath-sounds, diminished tactile and vocal resonance and absence of râles over this area. The heart borders and tones were normal. Liver, spleen, and kidneys were not palpable.

On October 5, 1927, aspiration of the chest was attempted over the right lower lobe. No fluid was obtained and the needle seemed to be obstructed by a hard mass which it was unable to penetrate. A grating feeling was felt and a drop of blood was present on the needle when it was withdrawn. A diagnosis of suspected lung tumor, most likely primary carcinoma, was made.

On November 16, 1927, the patient developed hoarseness and a laryngoscopic examination showed the cords apparently normal except slightly reddened.

December 20, 1927, patient developed a sudden hemorrhage and died. His weight on entrance, June 10, 1927, was 126½ pounds, and on November 5, 1927 it was 124½ pounds.

Laboratory findings: Repeated sputum examinations for tubercle bacilli were negative. Urine showed no albumin nor sugar. Blood Wassermann negative. Blood-count and differential were normal.

x-Ray examination, August 18, 1927, was negative for tuberculosis. Infiltrative density extended upward and outward from right heart border and right diaphragm, greatest in cardiophrenic angle. A few large air-vesicles were demonstrated in right cardiophrenic angle. A diagnosis of bronchiectasis was presumed from the x-ray findings.

On October 10, 1927, another x-ray was taken and reported as follows: Left lung and right apex were negative other than a few tiny glandular densities. An interlobular pleural line extended across much of width of right lung field below middle of length. Right diaphragm was obscured by shadow of not absolute density diminishing toward middle of lung field. A very few points of lesser densities were noted as cavities or small sections of greater degree of resolution. Resolving consolidation with pleural thickening is presumed.

Autopsy report, December 20, 1927.

Autopsy diagnosis: Primary bronchogenic carcinoma of the right main descending bronchus. Carcinoma metastases in the tracheobronchial lymph-glands. Blood-filled trachea and primary bronchi. Extensive ulceration,

gangrene and hemorrhage in the right lower lobe. Suppurative pneumonitis of the right middle lobe. Carcinomatous ulceration and partial erosion of the middle one-third of the esophagus. Carcinomatous thrombosis of the right pulmonary vein. Marked compensating emphysema of the left lung. Solitary calcified subpleural scar in the left upper pulmonary lobe. Pulmonary hyperemia and edema. Right obliterative fibrous pleuritis; marked cloudy swelling of the heart, liver, and kidneys; subacute splenic tumor; chronic fibrous perisplenitis; slight atherosclerosis of the aorta; hyperplasia of the mesenteric lymph-glands; blood-filled vasopharynx; slight cervical and inguinal lymphadenopathy; chronic fibrous periappendicitis.

Résumé: Carcinomatous thrombosis of the pulmonary vein; epithelial cell carcinoma of the lung; carcinoma metastases to the tracheobronchial lymph-glands.

**Summary and Conclusions.**—In making the diagnosis of primary carcinoma of the lung, it must be kept in mind that as compared with other lesions of the lungs, it is a very rare condition and every other possible disease should be excluded first. The acute and chronic infections, including syphilis, must be ruled out. The burden of proof lies in showing that there is not some other of the more common lesions of the lung present. A differential diagnosis must be made from pulmonary tuberculosis, fibroid phthisis, fibroid pleurisy, unresolved pneumonia, syphilis of the lung, mycoses of the lung, bronchiectasis, interlobular empyema, abscess of the lung, and enlargements and tumors of the mediastinum.

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## CLINIC OF DR. M. H. STREICHER

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### LINITIS PLASTICA (CIRRHOSIS OF THE STOMACH)

In spite of the vast amount that has been written and study that has been expended on linitis plastica, the fact remains that no case has ever been diagnosed clinically with reasonable amount of certainty. It is my aim to record a case which, while similar to the majority of reported cases in many points, showed an extraordinary dilatation of the colon in connection with the essential pathologic findings of linitis plastica.

**Case Report.**—E. H., a white female, fifty-two years of age, weighing 116 pounds, admitted to the Research and Educational Hospital of the University of Illinois, on August 26, 1926, presenting the following essential complaints: Pain in the abdomen of two years' duration, definitely related to meals and exaggerated after meals, constipation, nausea, vomiting, loss of 50 pounds in two years and generalized weakness. She states that she has had no trouble with her stomach until two years ago when she had an acute attack of nausea, vomiting one night after eating her supper; this attack subsided the next day and recurred several days later. Ever since the pain in the epigastrium has been constant and more severe—the vomiting spells more frequent, containing principally gastric contents but no blood.

**Past History.**—The patient had pleurisy and pneumonia when a child. Rheumatism at sixteen years of age. Tonsillectomy at thirteen years of age.

**Family History.**—Essentially negative.

**Physical Examination.**—Patient was anemic, wasted, appeared chronically ill. She weighed about 70 pounds, her best weight having been 116 pounds. The mucous membranes were extremely pale, the pharynx and tonsils anemic, and the tongue coated. The chest was poorly formed and wasted. The lungs showed impaired resonance over the right upper lobe and râles at the right apex. The abdomen was somewhat protuberant, an

obvious mass being present in the epigastrum, irregular in outline, hard to feel and not freely movable. The inguinal glands palpable and hard. Other physical findings were negative.

A tentative diagnosis of carcinoma of the stomach complicated with severe secondary anemia was made.

*Gastric Analysis.*—The vomitus was semisolid and liquid, of sour odor, and gave an acid reaction with litmus-paper. The

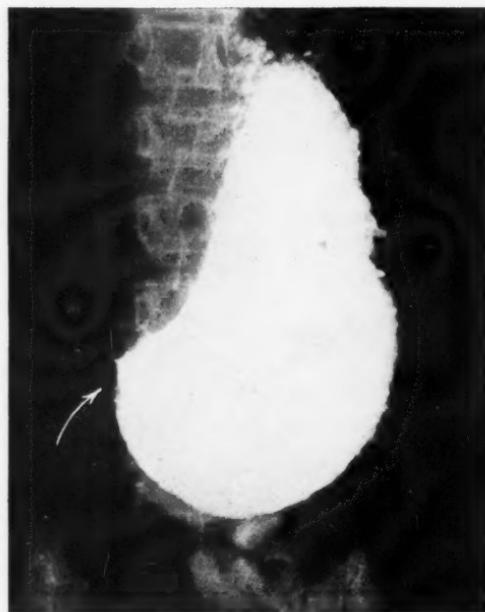


Fig. 52.—Linitis plastica. This diagram shows the stomach dilated due to pyloric obstruction. The arrow indicates the position of the pylorus.

amount of vomited material was greater than that of the test-meal, indicating retention. There was no free hydrochloric acid. The total acidity was 6 degrees; the test for lactic acid was slightly positive; the stomach contents were negative for blood, no Boas-Oppler bacilli found.

*Urinalysis.*—Several analyses were made on various days

during patient's stay in the hospital and were essentially negative.

*Blood-count.*—The red blood-cells numbered 3,430,000; white blood-cells, 5200; hemoglobin, 65 per cent.; polymorphonuclears, 80 per cent.; lymphocytes, 17 per cent.; large mononuclears and transitionals, 3 per cent.



Fig. 53.—Linitis plastica. Shows the stomach pictured in Fig. 52 after a posterior gastro-enterostomy.

The *Wassermann reaction*, with the cholesterin antigen, was 4 plus.

A *roentgenologic examination* made on September 16, 1926 showed that there was marked retention of the entire barium-meal and obstructive lesion at the pyloric end of the stomach. The heart and mediastinal structures were displaced towards the right. Numerous infiltrations were scattered throughout the right lung with some fibrotic strands suggestive of an old fibroid phthisis.

*Biopsy.*—One of the inguinal glands on microscopic examination was suggestive of a chronic non-specific inflammation.

In view of the degree of obstruction evident and the fact that the patient did not react to antiluetic treatment a laparotomy was advised.

*Operation.*—On September 30, 1926 the abdomen was opened by right rectus incision. The stomach was large, thick walled, becoming considerably thicker toward the pylorus. A hard,



Fig. 54.—*Linitis plastica.* Shows the rigid lumen of the stomach exposed. The arrow points to an adhesive fibrous band which extends from the omentum to the fundus of the uterus.

regular sausage-shape mass occupied the pyloric end, causing the obstruction. There were enlarged and indurated lymph-nodes about the lesser and the greater curvature. The glands were enlarged and red, but did not suggest malignancy. A gland removed from perigastric region was diagnosed as a chronic non-specific inflammation. A posterior gastro-enterostomy was done and patient was discharged from the hospital in fairly good condition.

Two months later patient came back to the out-patient department with recurrence of previous symptoms. On physical examination the abdomen was markedly distended and presented evidence of accumulation of gas. The x-ray of the gastro-intestinal tract showed a patent gastro-enterostomy and excessive accumulation of gas in the colon. About 3 inches from the external orifice the rectum presented an almost complete obstruction—hard, nodular, annular, not tender on palpation, and not bleeding on manipulation. Symptomatic treatment was instituted, but the patient grew progressively worse and expired on December 23, 1926. Fortunately permission for an autopsy was obtained.

#### **Autopsy:**

*Weight, 66½ lb.*

#### *Gross Pathology:*

*Stomach.*—The stomach was markedly diminished in size, firm, and when opened did not collapse. When cut it gave rise to a creaking and offered considerable resistance to the knife.

*Pylorus* was diffusely thickened and firm, lumen considerably narrowed, and the submucosa markedly thicker than normal. The gastro-enterostomy opening was patent. Transverse colon anterior to the stomach was much distended.

*Rectum.*—There was evident infiltration of the perirectal connective tissue with constriction of the rectum. The constricted area was 14 cm. long, 4.8 mm. thick; the thickness being made up mostly of submucosa and muscularis. No ulcerations present.

#### *Microscopic Pathology:*

*Stomach.*—The submucosa is very much thickened consisting primarily of dense fibrillar connective tissue.

*Rectum.*—Similar changes to those of the stomach were present.

*Remarks.*—The most interesting finding that perhaps explains the recurrence of symptoms of obstruction and also the abdominal distention is a band formed from the

large omentum, the lower end of which is attached to the fundus of the uterus with the colon on either side dilated.

*Anatomic Diagnosis.*—Carcinoma fibrosum—scirrhus (*limitis plastica* of Brinton) of the stomach with stenosis of the pylorus and of the rectum.

**Discussion.**—The French writers claim that all such cases are carcinomatous. Lyle (1911) speaks of a benign *limitis plastica* (diffuse fibrosis) and the malignant, calling attention to the fact that a clinical diagnosis of the benign form is rarely, if ever, made. In this case the positive Wassermann reaction and the pathology in the rectum simulating that in the stomach may be considered as specific etiologic factors and are associated.

The symptom of vomiting is of especial interest because of its underlying pathology. As the capacity of the stomach diminishes, the vomiting becomes more and more frequent—this point is given mention in the case reported by Singer (1926). This symptom was undoubtedly exaggerated in this case, due to the abdominal distention brought about by the band from the large omentum extending across the large colon.

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